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New Orleans Medical
and
Surgical Journal

$4.00 Per Annum, 35c Per Copy
Vol. 104, No. 2

AUGUST, 1951

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

SOME PRESENT DAY CONCEPTS OF CONGESTIVE HEART FAILURE AND ITS TREATMENT*

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Congestive heart failure is being revealed as a very complicated pathological state. The simple concept of a failing heart muscle and back pressure, with increase in blood volume due to retention of salt,[1] secondary to decrease in renal blood flow[2] or congestion of the kidneys will not alone suffice[3] without considering the more extracardiac disturbances. Sodium chloride retention,[4] which has received the greatest emphasis lately, has been suggested as being closely related to pituitary, adrenal, renal, and liver functions. The sodium and chloride blood levels, the carbon dioxide combining power, the matter of acidosis or alkalosis, the potassium and calcium depressions or elevations, nonprotein nitrogen retention, and probably the hemoglobin content, blood plasma proteins, especially the serum albumin levels, extracellular electrolyte concentration, and intracellular changes, all play a part. All of these factors are being suspected and investigated in the study of prerenal chemical disturbances in heart failure. These are all of importance in the elucidation, not only of the mechanism of edema formation, but also of its dissipation.

During the last two decades great progress has been made in the treatment of edema and congestive heart failure. In addition to adequate digitalization, more powerful and less toxic diuretics have been developed, salt intake has been restricted and absorption prevented, and the number of cases of refractory edema has been greatly reduced. Nevertheless, patients still die with slowly advancing relentless heart failure, which must be investigated and explained.

Most of the modern severe critics of the back pressure theory of congestive heart failure, accept the back pressure explanation for acute paroxysmal dyspnea, pulmonary congestion, edema, and frothy bloody bronchorrhea. This is admittedly acute failure of the left ventricle, which indicates failure of the drainage of the pulmonary areas, the shift of blood or plasma fluid mass from the greater to the lesser circulation. However, it is generally pointed out that in this acute condition there is no gain in weight or increase in total blood volume, or increase in venous pressure. All of these changes, however, are found in chronic heart failure in that there is an absolute increase in the total body fluid volume and blood plasma. This is a characteristic sign of chronic heart failure apparently responsible for the rise of venous pressure and engorgement of the liver.

The reasons for the pathophysiological changes are still not absolutely established. It seems logical to consider that the disor-

*Presented at the Seventy-first Annual Meeting of the Louisiana State Medical Society, New Orleans, May 8. 1951.

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ders of heart failure should begin to develop as a result of decreased efficiency of the heart muscle in most patients. It has been demonstrated[5] that reduction in cardiac output occurs in most cases of congestive heart failure, especially with low pulse pressure, as in coronary atherosclerosis, rheumatic myocarditis, diastolic hypertension, hypothyroidism, heart tamponade, pericardial constriction, erythremia and chronic severe dietary deficiency. However, there is another group of conditions in which the picture of congestive heart failure appears and the cardiac output is usually above normal in high pulse pressure conditions such as in syphilitic aortic regurgitation, systolic hypertension, hyperthyroidism, arteriovenous shunt, congenital lesions, anemia, and beriberi. In these we have been prone to consider that there has been a relative drop from a high level to levels still above normal, or to just normal levels of cardiac output. The cardiac output drop to a relatively low or to an actually low level seems to affect the kidney function, especially to reduce glomerular filtration. The stress of reduced circulation and anoxia affects other essential organs, especially the pituitary, adrenal, and thyroid glands. It has been repeatedly demonstrated in heart failure cases that the sodium excretion and the day volume is decreased, while the night volume is increased.

A clear understanding of the mechanism of sodium chloride and water retention in heart failure cases may be elucidated by a study of the factors that control sodium chloride and water excretion in urine of normal individuals. In normals the diurnal excretion of salt and water is greater than the night output. These outputs are not dependent upon changes in filtration or renal blood flow. Increase and decrease in salts and water in the urine are under the control primarily of tubular reabsorptions.

Exercise, sitting, standing, as well as stepping produce a drop in sodium chloride. excretion. Restricted sodium chloride intake decreases sodium chloride in the urine and the extracellular fluid volume, while excessive sodium chloride intake increases sodium chloride in the urine and the extracellular fluid volume. Hormones of menses, ACTH, cortisone, testosterone, and desoxycorticosterone acetate increase sodium retention. Pitressin decreases water output but sodium chloride total excretion remains the same. Concentrated sodium chloride intravenously causes water and salt excretion that is refractory to pitressin. Alkalosis produces excessive sodium excretion. Acidosis causes excessive chloride excretion.

Chronic congestive heart failure is characterizes by a retention of sodium chloride and water during the day with a rise in the output at night that is however not fully compensatory. This is just the opposite from normals; otherwise the congested cardiacs' reactions to exercise, infection, anoxia, and hemorrhage are similar to normals but generally dampened.

Glomerular filtration of sodium chloride is usually reduced in congestive heart failure patients. At the same time in congestive failure there is renal tubular avidty for sodium, and sodium retention is conspicuous. Retention of sodium may be then considered to stimulate elevation of the blood volume and of the venous pressure. A stress factor elaborating salt retaining hormones may be operative in part. In experimental animals a rise in the venous pressure in the kidney decreases sodium chloride output without a change in fltraton or renal blood flow.

The traditional concept is that high venous pressure produces a high hydrostatic capillary pressure in the venous limb which overcomes the onconic or osmotic pressure of plasma proteins and thus prevents the return of sodium chloride and water to the vascular compartment from the interstices.

Prerenal deviation of sodium chloride and water immediately into tissue interstices occurs in hypoproteinemia. It has been stated that such a mechanism would cause a decrease in the blood volume and thus not be a part of the mechanism of congestive heart failure in which the plasma

volume was increased. Then, too it has been pointed out that patients who have had congestive failure continue to have difficulty in handling sodium chloride after the venous pressure has returned to normal.

Noncardiac conditions such as infections, and hemorrhage have shown temporary sodium chloride retention. In cardiacs the defect persists for years even when there is no increased venous pressure. Qualitative along with quantitatve differences have occurred as a result of a renal nocturnal excretion.

In recent years there have been demonstrated disturbances in the electrolytes which sometimes may be controlled and reversed, but which if not recognized lead to dissolution. Just what is responsible for the death from pure heart failure is not known. It may be in the irreversible electrolyte upsets that may come on spontaneously in morbid states. They are more frequently precipitated in a patient with congestive chronic heart failure by the too vigorous application of mercurial diuretics. The development of more potent mercurials and the additional restriction of salt intake and uptake by resins contribute to subnormal electrolyte levels. Frequent repetition of mercurial diuretic injections at intervals of twelve to twenty-four hours may produce the effects of sodium chloride and disturbed potassium and acid-base equilibrium. These effects may persist and become accumulative, producing hyponatremia, hypochloremia or both in a serious salt depletion syndrome and usually a refractoriness to further mercurial diuresis. Sometimes glomerular and tubular pathology are aggravated.

Refractory edema in persistent heart failure calls for a complete survey of the whole situation. If and when a mercurial diuretic fails to produce a diuresis, it is necessary to make a study of the blood electrolyte levels, at least the sodium and the chloride independently, and the carbon dioxide combining power. It may be a sodium depletion acidosis or chloride depletion alkalosis which must be reversed. It is important to determine how much actual restriction there was in the salt intake, whether effective amounts of ammonium chloride were being given, whether digitalization has been complete and maintenance dosage has been sufficient, and whether the physical activities have been kept at an absolute minimum.

It is necessary to rule out kidney or liver disease that may have produced a hypoproteinemia or hypoalbuminemia, severe anemia, and mechanical factors such as constrictive pericarditis and tricuspid stenosis that may cause liver engorgement, ascites, and edema. Active rheumatic fever or any other acute infection that produces fever or causes a reversible heart disease as arteriovenous aneurism, myxedema, beriberi should have been treated or corrected before concluding that the patient is in a terminal stage of refractory heart failure.

Successful therapy consists in a judicial combination of methods to correct any cardiac, prerenal, general, or local factors that contribute to an edema formation or its persistence. Our efforts are no longer directed toward getting more potent mercurial diuretics. In the last few years we have been more concerned with effective but less toxic drugs. Low sodium diets have been devised and many patients take these without much protest. Yet it is difficult to prepare salt free diets in the home. By exchange of resins, particularly those with potassium added, it has been possible to get results without being too stringent about salt intake but potassium and calcium deficiencies may develop. It has been shown that mercurial diuretics may produce a hypochloremia alkalosis with normal sodium and refractoriness to the drug. On the other hand, primary or secondary depletion of sodium and acidosis seems also to be responsible for the refractoriness. Complete chemical studies are necessary to establish the situation and to reveal any other factors which may be present.

We have in recent years continued our studies on the effect of diuretics on urine output and on body weight, blood volume and venous pressure, sodium chloride, po-

tassium and calcium clearances. More recently we have determined glomerular filtration by inulin clearance and total renal flow by para-aminohippurate clearance in the hope of getting the evidence as to the mechanism of action of digitalis, aminophyllin, and thiomerin, and whether or not damage has been done during diuresis. We have further hoped that this might throw some light on the mechanism of accumulation of edema fluid in patients with congestive heart failure.

Congestive failure patients show mostly a qualitative long persistent sodium retention, and slower excretion of sodium. There is characteristically a relative rather than an absolute inability to excrete sodium chloride. Just as in normal individuals there is retention of sodium chloride during exercise, infection, and hemorrhage, but this occurs in a more exaggerated form or to a greater degree in cardiac patients.

The complete elucidations of the mechanisms of salt and water excretion in heart failure required still further observations in the normal and abnormal states. The studies of the group at the Southwestern Medical School have shown that the increased excretion of sodium chloride on sitting, standing, and stepping, in hemorrhage, and in salt overloading may be inhibited by putting a venous obstructing band about the neck. All of these conditions had caused only moderate decline in the excretion of sodium and the reduction could be prevented by compression of the neck. The excretion of chloride tended to parallel that of sodium. Bleeding seemed to cause a rise in the potassium excretion in some instances. These findings furnished to Harrison and his group present evidence supporting the concept of a homeostatic mechanism generally or a center in the brain. This homeostatic mechanism is concerned with sodium conservation, and is apparently affected by alterations and by distribution of blood within the body. The authors felt that their data did not support the idea that a decline in cardiac output constituted the stimulus to the retention of sodium chloride.

In congestive heart failure conditions it was found that acidosis with ammonium chloride produces excess of chloride excretion. Alkalosis produces excessive sodium excretion. It is most important to determine what produces a balanced excretion of sodium and chloride with neither in excess of the other.

Renal hemodynamic disturbances correlate with the reduced cardiac output concept of heart failure. When glomerular sodium chloride filtration is lowered without corresponding decrease in tubular reabsorption, edema results. On the other hand, in patients with glomerular nephritis tubular reabsorption is defective, edema may not result as the sodium is excreted. Changes in the sodium excretion can be produced in patients with congestive heart failure to the extent of the change governed by the amount of sodium filtered. Restriction of sodium chloride intake causes edema to clear up even though the filtration rate remains low. Bed rest dissipates massive edema unless filtration is markedly reduced. In patients with heart failure exercise causes great retention of sodium and water, and massive edema, while in normals, exercise produces only slight sodium retention.

Reduced cardiac output and elevated venous pressure definitely affect renal function. The role of the kidney has superseded physical osmotic capillary pressure mechanism in the production of edema. Other prerenal factors may participate in the diversion of the sodium and chloride and water to tissues. Reduced blood flow may also affect the important endocrine secretions from the pituitary, adrenal, and thyroid glands.

Distribution of orally ingested salt and water depends on local factors, hydrostatic inverse capillary and tissue pressures. Injection intravenously of salt solution produces pulmonary edema, especially in the presence of some left heart failure. In right ventricular failure there is less tendency to pulmonary edema; however, it causes edema of the dependent parts.

THE MECHANISM OF MERCURIAL DIURESIS

Mercurial diuresis has generally been considered to be effected by the decreased reabsorption of sodium and water by the tubular epithelium. Richards[32] observed that in the frog kidney diuresis was due to decreased rate of tubular reabsorption and that there was no increase in glomerular filtration. Schmitz[33] studied the effect of salyrgan on dogs and reached similar conclusions. Herrmann, *et al*[34] found that salyrgan produced a striking effect upon the tubular epithelium of patients in congestive heart failure and accomplished diuresis primarily by defective tubular reabsorption. Christian and Bartram[35] showed that mercury acts on the kidneys alone. Quite recently it has been postulated by Duggan and Pitts[36] that organic mercurial diuretics decrease the reabsorption of sodium in the distal tubules. Our patients demonstrated that the mercurial depressed the tubular sodium reabsorptive mechanism and we have recorded great outpouring of sodium and water after the injection of Thiomerin (See Fig. 1).

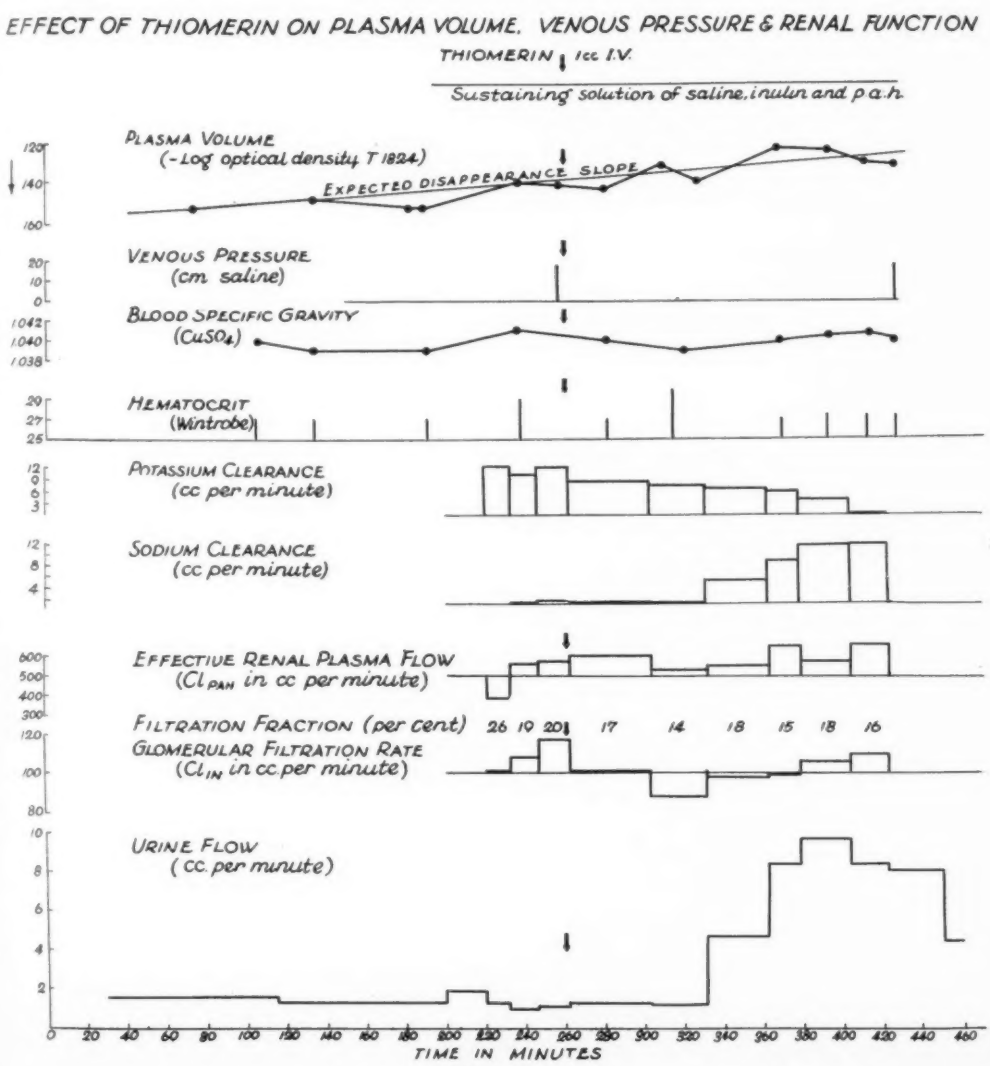

Figure 1

An initial hemodilution of the blood during mercurial diuresis has been observed by some investigators. It is suspected that this was due to the theophylline combined in the diuretic. Schmitz[33] noted no such effects in his studies. Hemoconcentration during diuresis was noted by Bryan[39] and his associates who found no constant change in specific gravity or colloidal osmotic pressure immediately after injection of mercury. In our laboratory we[40] found in general a hemoconcentration with mercurial diuresis. In more recent studies we have noted that venous pressure and blood volume changes are secondary to the loss of sodium and water that results from mercurial diuresis.

Herrmann *et al* have maintained that immediate increases of sodium excretion and a very transient lowering of serum sodium is the trigger mechanism which calls sodium and water from the tissues after in-

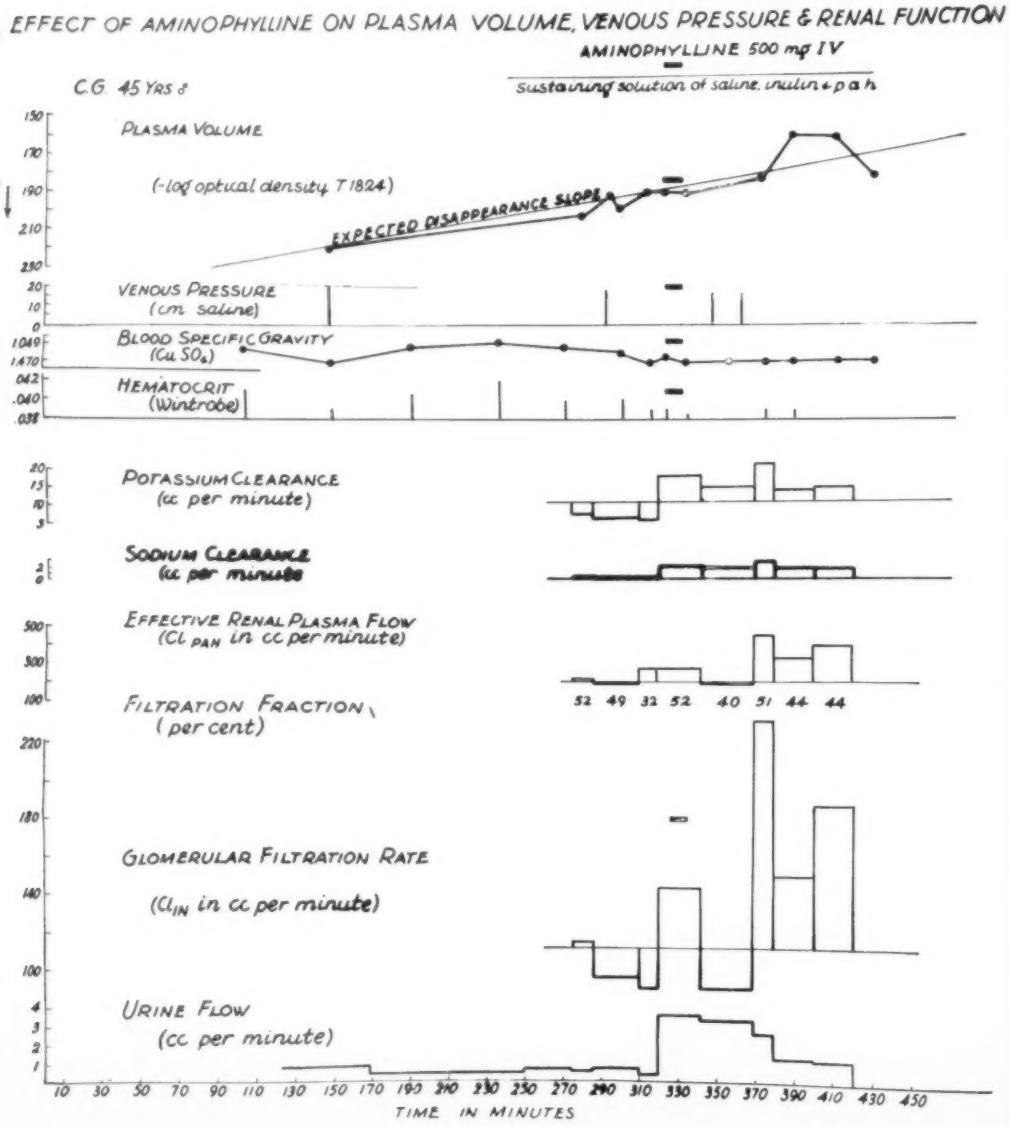
EFFECT OF AMINOPHYLLINE ON PLASMA VOLUME, VENOUS PRESSURE & RENAL FUNCTION
AMINOPHYLLINE 500 mg IV
sustaining solution of saline, inulin + p a h
C.G. 45 YRS ♂
PLASMA VOLUME
(-log optical density T 1824)
EXPECTED DISAPPEARANCE SLOPE
VENOUS PRESSURE (cm. saline)
BLOOD SPECIFIC GRAVITY (Cu SO4)
HEMATOCRIT (Wintrobe)
POTASSIUM CLEARANCE (cc per minute)
SODIUM CLEARANCE (cc per minute)
EFFECTIVE RENAL PLASMA FLOW (Cl PAH in cc per minute)
52 49 32 52 40 51 44 44
FILTRATION FRACTION (per cent)
GLOMERULAR FILTRATION RATE (Cl IN in cc per minute)
URINE FLOW (cc per minute)
TIME IN MINUTES

Figure 2

jection of a mercurial. More refined methods will be required to establish this belief.

MECHANISM OF DIURESIS WITH AMINOPHYLLINE

The mechanism of diuresis with aminophylline is still controversial. Evidence presented by Schmitz and Herrmann's group suggested that aminophylline acted by increasing glomerular filtration by augmenting glomerular circulation. Until recently we felt that any defect in tubular reabsorption was due to a secondary circulatory effect incident to increased blood flow through the tubular plexuses and the increased rate of passage of glomerular filtrate through the tubules interfering with reabsorption as the filtrate rapidly passed the epithelial cells.

We have noted increased sodium excretion by all of the patients to whom aminophylline was given, as shown in Figure 2. In some of our patients there was apparent hemodilution immediately after giving aminophylline; in others hemoconcentration appeared to occur. Regardless of the changes in concentration of T-1824 attributable to aminophylline sodium excretion was significantly enhanced. It would appear that aminophylline exerts a renal effect which is independent of its general circulatory effect.

The nature of the change in circulatory dynamics which produce such profound shifting changes in dye concentration as noted in some patients is not clarified by our studies. The temporary fall in concentration of T-1824 may be a peripheral vascular phenomenon. McMichael *et al*[16] have suggested that aminophylline reduces venomotor tone, in addition to its stimulating effect on the myocardium. Venodilatation may result in a fall in hydrostatic pressure so that there is temporary rush of edema fluid from the tissue interstices into the veins causing the transient hemodilution noted. Further studies are in progress to compare plasma volume shifts occurring in patients when they are in failure and when cardiac compensation has been restored in them.

SUGGESTED MANAGEMENT OF CONGESTIVE HEART FAILURE

The excretion of sodium is increased by all of the diuretics. The behaviour of the mercurials and aminophylline is alike in effecting an early rise in sodium excretion. With digitalis there is apparently a slower but more persistent rise in urinary sodium concentration and output.

In the management of congestive heart failure prompt determination of the venous pressure will provide a firm base line for evaluation of treatment to be undertaken. When there is marked elevation of venous pressure, or a sudden rise in venous pressure as in acute left ventricular failure, slow intravenous administration of aminophylline is the primary treatment of choice. It is in effect a bloodless phlebotomy. While the effect of aminophylline is being dissipated, digitalization should be carried out, thus perpetuating and augmenting lowered venous pressure produced by aminophylline.

The routine use of intravenous digitalis is not recommended and is often undesirable; one dose digitalization intravenously, was carried out by us only to study the effects of the drug and because the maximal effect of digitalis is not apparent when half-digitalizing doses are employed. Numerous preparations available are capable of inducing full digitalis effects in a few hours when given by mouth. If digitalis, preceded by aminophylline, does not appear adequately to mobilize the sodium and water, thiomerin is in order in the management of the edematous patient.

SUMMARY

1. Mercurial diuresis results in a prompt increase in sodium and water excretion by depressing tubular reabsorption. The plasma volume and venous pressure gradually fall in response to diuresis.

2. Aminophylline also produces a prompt rise in sodium and water excretion. This renal effect may or may not be associated with a general circulatory effect. In the patient in congestive failure, aminophylline appears to produce diuresis by increasing glomerular filtration rate and effective renal plasma flow.

3. Digitalis in full digitalizing doses given intravenously effects a prompt fall in venous pressure. There is a slower onset of diuresis but sodium excretion is augmented as well as water excretion.

4. Aminophylline seems to be the drug of choice when a prompt lowering of venous pressure is desired. Digitalis may next be employed to maintain the lowered venous pressure, further diuresis, and for cardiotonic effects. Thiomerin for its action on the renal tubules, may be employed if aminophylline and digitalis do not restore circulatory equilibrium in the grossly edematous patient.

5. In refractory cases, the whole situation must be resurveyed and restudied as to diagnosis, and adequacy of therapy, especially digitalization. If mercurial diuretics are ineffective and if weakness or renal changes have developed, aminophylline should be tried. However, the electrolyte balance must be studied with blood nonprotein nitrogen, carbon dioxide combining power, chlorides, potassium, and calcium must be determined, and compensatory steps taken according to the defects found.

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CANCER OF THE RECTUM; ITS RECOGNITION AND TREATMENT*

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The surgical attack on the malignancy of the lower gastrointestinal tract has during the past two decades kept pace with surgical progress elsewhere. This is exemplified by marked improvement in mortality statistics, in morbidity and in operability rates, despite the fact that patients continue to appear with cancer of the rectum and colon at the same long interval after the initial symptoms should have been recognized.

It is trite to repeat the continual warnings of the sentinel symptoms but the very fact that some forty to fifty thousand individuals die yearly of cancer of the colon and rectum and two or three times that many have the disease is ample reason for repetition even though it be platitudinous. It is a deplorable fact that cancer of the rectum, which may always be diagnosed, is passed up by the patient and the doctor frequently to a point where it has developed to an advanced stage and even metastasized widely before a diagnosis is made. Patients so frequently hesitate to seek advice and examination and doctors so frequently are cursory in their examination that our figures of cures will probably not be revised downward soon except as a result of repetition, admonition, and urgent propaganda.

Seventy per cent of the cancers of the colon occur in the sigmoid and rectum and are diagnosible by digital examination or proctoscopy or both and with or without biopsy. Interestingly enough, in this region of the large bowel about 70 per cent of the polyps occur and this is a distinct facet upon which to build a thesis concerning the etiology of rectal cancer, but more about that later.

There have been a number of trends which have developed in the surgical treatment of cancer of the lower gastrointestinal tract in recent years—and I take it no one will be offended at the statement that surgery is the only treatment for cancer in this location.

In the past ten years the following marked changes have become more or less standardized in the hands of all experienced surgeons dealing with lesions in this location.

1. More attention to the preparatory period preliminary to operation.

2. The recognition that cleansing of the bowel is as essential a part of the program as is the operation itself.

3. The lavish use of whole blood in the preoperative period.

4. Use of antibiotics and other chemotherapeutic agents in the reduction of the bacterial content of the bowel itself.

5. The widespread use of single stage maneuvers.

6. The controversial question of the preservation of the sphincteric mechanism and/or the establishment of a low primary anastomosis operation following the resection.

SYMPTOMS

The symptomatology of cancer of the rectum revolves around: (1) Blood in the stool or on the stool; (2) change in bowel habit. Strangely enough, the early symptoms which are so essential to observe are either masked or ignored or misinterpreted. It is not possible to determine the exact time at which cancer of the rectum begins, but it is highly possible that most of them have existed many months or even a year or more prior to their recognition. Most surgeons believe that between nine and twelve months elapse prior to diagnosis, and that has been my experience in a large series of cases.

Bleeding in cancer of the rectum means bright red blood and bright red blood more often than not is due to some other lesion, such as hemorrhoids, fissure, and fistula, rather than to cancer. The blood may be mixed with the stool or simply streak the stool and it should be remembered that it

*Presented at the Seventy-first Annual Meeting of the Louisiana State Medical Society, New Orleans, La., May 8, 1951.

does not appear until the mucous membrane has been eroded and that may be months from the initiation of the cancer. On occasion, a sudden hemorrhage may call attention to the presence of the growth. Small amounts of blood on the paper are among the earliest symptoms in most cases.

Change of bowel habit often is urged as among early symptoms and this simply means alternating periods of diarrhea and constipation and it should be noted that these periods are of short duration usually. Constipation is a cardinal complaint in about two thirds of the cases and alternates with a psuedo-diarrhea which may mean two or three loose stools daily. Frequent stools often follow the taking of cathartics, and therefore, are not connected with diarrhea. The so-called ribbon stool has no significance as a symptom of cancer of the rectum since it depends upon the consistency of the stool and the tonicity of the anal sphincter and is seen more often in constipation than in organic diseases of the rectum.

Pain is seldom a symptom of cancer of the rectum until the malignancy is far advanced. When the growth has become fixed with involvement of neurological elements severe pain may result, but usually pain occurs only in the late cases.

Excessive loss of weight and strength are worthless symptoms in the early recognition of this condition. Obviously all or any of these symptoms are inadequate factors by which to diagnosis cancer of the rectum and their main usefulness is to succeed in calling attention to some unusual condition in the bowel bringing about its detection in the course of examinations more thorough than usual. In the presence of these suggestive phenomena it is therefore imperative if an early diagnosis is to be made that one resort to the simple expediency of digital and proctoscopic examination. By the former alone a presumptive diagnosis is possible in more than 90 per cent of all cancers of the rectum and by proctoscopic examination all carcinoma of the rectum and sigmoid up to 25 cm. may be accurately diagnosed; albeit, occasionally a biopsy is necessary for absolute confirmation. While it is my custom to biopsy and grade all cancers of the rectum and lower sigmoid I believe that proctoscopic examination and palpation will be sufficient in practically all cases to make an accurate diagnosis.

I would emphasize that roentgenologic examination for the diagnosis of rectal and low sigmoid growths is not indicated nor is it accurate in a large number of cases because of the bony pelvis which prevents satisfactory examinations. Cancer of the rectum can be accurately diagnosed in all cases by digital and proctoscopic examinations plus biopsy and to wait until the appearance of classical textbook symptoms such as loss of weight, strength, profound anemia, dehydration, and pain, is merely postponing the diagnosis until generalized metastasis has occurred and there is no hope of a cure.

TREATMENT

Treatment of cancer of the rectum, with few exceptions, remains surgical and the more radical the surgery, with or without a colostomy, the better the end results. All surgical treatment, of course, should be preceded by an adequate preoperative period of preparation, the objects of which are:

1. Decompression and cleansing of the bowel.
2. Replacement of blood loss and the establishment and maintenance of protein balance by transfusion of whole blood.
3. Steps to combat dehydration with glucose and saline solutions.
4. Concentration of vitamins, especially vitamins C and K.
5. The use of chemotherapeutic agents.

These objects are best carried out in a hospital over a period of four to six days, but there are exceptions to this and it is entirely possible that many cases may be prepared in an ambulatory manner without hospitalization.

The rehabilitation which thus combats anemia, insures against electrolytic imbalance and dehydration, restores liver glycogen and vitamin reserve, while at the

same time, replacing protein deficiency is of vital importance and cannot be overemphasized.

The cleansing of the bowel by purgation and rectal irrigation is of no less importance and should be persisted in with considerable vigor if there is chronic obstruction due to a local growth.

As for the use of chemotherapeutic agents and antibiotics, I must confess that while I was dilatory in accepting them as of great value, I am forced to the conclusion that their use is of great importance in preliminary preparation by sterilizing the bowel content and thus aiding greatly in the prevention of postoperative infection.

What agents one uses seems of not too great importance and we go from sulfathaladine to terramycin to chloromycetin, to some new drug, with considerable rapidity which is satisfactory evidence of the potency of all of them and which I think should convince us all of their effectiveness.

CHOICE OF OPERATION

Trends in the treatment of cancer of the rectum have been not too numerous in the past decade save for the tendency of a few surgeons to press the idea of sphincteric saving operations at a bold rate and, what seems to me, an unwarranted extreme. In this view I am supported by many of the most experienced surgeons operating upon rectal cancer and it seems difficult to combat the statistical evidence which large series of cases offer. It is a well known axiom that the lower down in the gastrointestinal tract the growth occurs, the less satisfactory the prognosis, and while I am convinced that sphincter saving operations have their place, particularly as palliative procedures, I am equally sure that growths which occur below the reflection of the peritoneum should certainly be operated upon by a radical one-stage combined abdominoperineal operation after the technique of Miles or some variation of his procedure. The choice of operation lies among the following:

1. Combined abdomino resection in one stage (Miles).
2. Colostomy and posterior resection in two stages (Mummery).
3. Local resection without colostomy.
4. Operations preserving the sphincteric mechanism.

In the average individual, who is a reasonably good risk and whose growth lies below the peritoneum, I believe Miles' operation is unquestionably the procedure of choice. It can be done with a very satisfactory mortality, a satisfactory morbidity, and it excels in results. In the last 107 cases which I have done by Miles' technique there have been 3 deaths or a mortality rate of 2.7 per cent. The resectability rate was 81.8 per cent.

Occasionally, one finds an individual in whom, because of co-existing debilitating disease or obesity or some other good reason, the Miles operation seems better supplanted by a graded procedure. Here Mummery's operation is of value. Into this same category falls the local excision of low lying growths without a colostomy, in an occasional case. I think that any operation which gets rid of the cancer in a debiliated individual, with a high risk, is worth-while, if it is a success, and occasionally satisfactory results do accompany these local procedures.

As for saving the sphincteric mechanism or doing anastomoses low in the pelvis, I feel that gradually these procedures are being more and more stabilized, and furthermore, that the position I assume; namely, of drawing a line at the peritoneal reflection below which an anastomosis is not made, is becoming more and more popular. It is not a particularly difficult feat for an average technician to do an anastomosis low in the pelvis, but the crux of the situation seems to me to lie in the question as to whether or not more people will be saved by doing radical excision with a colostomy than by doing a low anastomosis. I have no hesitancy in urging colostomy on people whose growths are below the peritoneal cavity and I shall continue to feel that the position certainly is a safe one until more convincing data is assembled to disprove it.

On the other hand there are many inoperable growths, because of liver metastasis or peritoneal implants, which may be removed locally, and in them I think it is perfectly reasonable to do anastomosis, for unquestionably, the individual will be more comfortable by preservation of the sphincteric mechanism than with a colostomy.

All of this adds up to the fact that no single type of procedure fits all cases of cancer of the rectum. Indeed, here as elsewhere, flexibility of application must be a cardinal principle and one should never hesitate to do an occasional operation which varies from the ideal if by so doing life will be prolonged or comfort given to the patient. Unquestionably, too little attention has been paid to doing palliative procedures when the growth is removable but when distant metastases obviously rule out a cure. Certainly an individual dies of cancer of the liver, if the growth has been removed, much more comfortably than if the growth has been allowed to remain and extend locally to involve neurological elements and other viscera.

In all clinics over the past two decades there has been a remarkably satisfactory lowering of surgical mortality and morbidity with very definite increase in five and even ten year freedom from recurrence. How much further one may extend radical surgical procedures advantageously becomes a question, but certainly a review of long term freedom from recurrence emphasizes the fact that continued efforts to extend our field of operation should be persisted in.

PROGNOSIS

A review of any series of cases shows a remarkably satisfactory curability rate when compared with cancer elsewhere in the gastrointestinal tract. Recently, Graham and I have circularized a number of clinics and published the results of this survey. There is a notable difference in the curability rate of private patients in comparison with ward patients in all clinics. For this there are many specific reasons which need not be gone into here.

Dating back to the work of Daniel Fisk Jones in 1936, we find the survival rate in his cases to vary from 52.6 per cent to 61.5 per cent—five years' survival. His operability rate was 63.5 per cent.

In 1948, with Johnston, I published a series of 162 combined abdominoperineal resections with a three to four year survival of 63 per cent, and a four to five year survival of 56 per cent. The mortality rate in this series was 5.3 per cent, the resectability rate was 75.1 per cent. This trend toward elevation of the resectability rate and decline of the mortality rate has increased no little over recent years.

One stage operations have gradually replaced in large measure graded procedures and one must be in accord with Jones who said, "I have gradually increased the number of one-stage operations and decreased the number of two-stage operations and believe that this should be done as men find their ability to do the one stage operation increasing. I still feel that there are a few cases in which I want to do and which are not fit for a one-stage operation." That this is a distinct advance is indubitably true and it does not sacrifice, but rather increases the scope of the operative maneuver.

Another point is that small and supposedly early cancers of the rectum should be invariably subjected to the most radical type of extirpation. Indeed some of the smallest cancers are high grade and it is not an uncommon thing to find liver metastases and general peritoneal involvement in cancers which have given few or no symptoms and which are small punched-out ulcers, but on biopsy show grade 3 or 4. There is no substitute for radical surgery in dealing with cancer of the rectum and it should inevitably be pressed within reasonable limits of mortality.

On the other hand the encouragements which one receives in reviewing statistical data from large groups of cases emphasize the point that no where else in the gastrointestinal tract are ultimate end results comparable to those received from surgical attack on cancer of the colon or rectum.

Nowhere else does the five year freedom from recurrence compare statistically with that from operations for cancer in this location. Such encouragements, of course, should stimulate continued efforts to extend operative procedures until such time as a simple test for determining the presence of a cancer in its early stages is developed and a different type of treatment evolves therefrom.

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NONTRAUMATIC RUPTURE OF THE COMMON BILE DUCT*

TWO CASE REPORTS

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Since nontraumatic, spontaneous, rupture of the common bile duct is rare, it would seem that every authentic case should be reported. Ruptures of the common bile duct have been reported from time to time and the clinico-pathological features presented. The etiology may be associated with calculi, acquired or congenital stricture of the common bile duct, malformations (congenital cystic dilatation), infection and tumors.

The general incidence of common duct perforations relative to the remaining portion of the bile duct system may be appreciated in the figures of McWilliams.[1] In a survey of 90 cases of spontaneous rupture of the biliary tree, only four (4.4 per cent) were found in the common duct. Apparently, these cases were unassociated with previous operative procedures and calculi were present in the common bile duct. Unfortunately, detailed information was not presented.

In 1930, Bailey[2] recorded a case of spontaneous perforation of the common bile duct in a 45 year old male. At operation, the gallbladder appeared normal, although it contained small calculi. A probe was passed through the ducts but failed to reveal obstruction or a definite perforation, but at autopsy a point of rupture was located at the junction of the cystic and common ducts in a posterior position.

In 1932, Vale and Shapiro[3] reported a nontraumatic perforation of the common bile duct in a 23 year old Mexican woman. At operation, bile was found in the peritoneal cavity. The gallbladder was chronically inflamed and contained innumerable calculi, which were removed. A definite site of perforation was not found. However, a posterior perforation may have been present. The patient had a peritonitis but slowly recovered to a state of good health.

In 1941, Newell[4] recorded a case of bile peritonitis incident to an acute spontaneous rupture at the common bile duct in a 63 year old man. He contended that no cause could be determined for the perforation. The common bile duct was four times normal size and had assumed the functions of an atrophic gallbladder with consequent dilatation and choledochitis. Probably, the inflammatory process was followed by an acute exacerbation with edema or spasm of the sphincter of Oddi, resulting in increased intraductal pressure and rupture at the weakest point. The possibility of a minute calculus embedded in the wall of the common bile duct was considered. This patient recovered.

Additional cases have been recorded by Traube,[5] Wernsdörfer,[6] Dijkstra,[7] Gurevich,[8] LeClerc,[9] Maitre[10] and Ferrancani.[11] As previously indicated, the above cases were unassociated with previous operative procedures. Following surgery, only a few cases of spontaneous rupture of the common bile duct have been recorded.

In 1935, Wolfson and Levine[12] recorded 3 cases of spontaneous rupture of the common bile duct as a sequel of choledochostomy. In these cases, the time interval between the two operations was one month, two months, and two and one-half months. Recognition of the possibility of perforation of the common bile duct following choledochostomies will permit immediate operative intervention. In this type, the etiology is associated with a necrotizing inflamma-

*Presented at the Seventy-first Annual Meeting of the Louisiana State Medical Society, New Orleans, May 8, 1951.

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tory process at the site of the choledochostomy.

In 1938, Newburger[13] reported a case of spontaneous rupture of the common bile duct incident to stones that were overlooked at the original operation, ninety-six days prior to the perforation. Post-mortem examination revealed a perforation of the stump of the cystic duct and three black pigmented stones impacted at the junction of the hepatic, cystic, and common ducts. These calculi, each 1.5 cm. in diameter, were faceted, lying in pocket dilatations, so that one stone was in each duct. The bile duct system was definitely dilated. He further reported a review of the literature with 12 case reports, all following surgery, 8 of which were substantiated by operation or autopsy.

Plimpton and Clagett[14] of the Mayo Clinic reported 1 case of spontaneous rupture occurring seven months after an operation on the biliary tract. The first operation was a cholecystectomy in 1937, performed because of jaundice. In 1941, a second operation was performed for a stricture of the common bile duct. The final operative procedure, in 1942, revealed a small amount of free bile in the peritoneal cavity and a large space containing approximately 2 liters of bile which was entered after freeing the edge of the liver from the anterior abdominal wall. Owing to the precarious condition of the patient, exploration was not carried further. Adequate drainage was established and the incision was closed. The patient made an uneventful recovery. Although the actual site of perforation was not determined, it was thought that it occurred either in the stump of the cystic duct or the site of the choledochostomy.

In 1943, Brunschwig presented 2 cases of postcholecystectomy rupture of the common bile duct. In the first case, the patient was a 67 year old female, who had a cholecystectomy for calculi. Two months later, a cholecystectomy was performed; the common bile duct contained a calculus, which was removed. Approximately a year afterwards, the patient returned with right upper quadrant pain, icterus, nausea, and vomiting. Necropsy on the seventh hospital day disclosed a perforated choledochus, 3.5 cm. below the cystic duct and a single faceted cholesterol stone, distal to the site of rupture which measured 1.4 cm. in diameter. In the second patient, a female, age 72, a cholecystectomy had been performed a year previously. Recurrence of gallbladder-like symptoms resulted in an exploratory laparotomy. Two centimeters above the duodenal margin, there was a 1.5 cm. perforation in the common bile duct associated with a 1.5 cm. stone near the duodenal portion of the choledochus. Proper operative and postoperative procedures resulted in a complete recovery of this patient.

In 1947, Drieling[16] recorded a case of spontaneous rupture of the common bile duct following choledocholithotomy. The operative procedure had been performed six months before the rupture. Exploration disclosed the perforated duct and a large round calculus embedded in its distal portion. After a rather long convalescence, the patient was reported in good health. Drieling emphasizes the importance of common duct stone in postoperative rupture which may be associated with spasm of the sphincter of Oddi. According to this author, there are only 27 case reports of postoperative rupture of the choledochus.

Additional cases have been presented by Masciottra,[17] Schen,[18] Durst[19] Bernard,[20] Mirizzi,[21] and Poppovici.[22]

Traumatic rupture of the choledochus is a very rare entity. In 1948, Hicken and Stevenson[23] reported a case wherein a tractor crushed the abdomen of a 7 year old boy. Exploratory laparotomy disclosed a laceration of the posterior wall of the common bile duct. Adequate drainage and supportive therapy was followed by complete recovery. These authors stress two valuable points.

1. If the extrahepatic biliary system is completely decompressed, laceration of the common bile duct will heal spontaneously.

2. Operative cholangiograms should be commonly employed to accurately localize

lacerations or perforations in the biliary tract.

These statements are worthy of our sincere attention.

Our 2 cases of nonoperative rupture of the common bile duct follow:

CASE REPORTS

Case No. 1. White female, age 45, admitted November 6, 1948; expired November 11, 1948.

Chief Complaint: Right upper quadrant pain.

Present Illness: The patient had been in fairly good health until three days before seeking medical relief, when she noticed soreness and dull pain in the right upper quadrant. This pain, colicky in type, gradually became more severe, especially at night. It remained in the right upper quadrant; no radiation was noted. There was nausea but no vomiting. The urine was dark but the stools were normal. No chills, fever, or jaundice were noted. Since the onset of the illness, the patient had been slightly dyspneic.

Past History: The patient had had recurrent attacks of right upper quadrant pain for six years, the frequency, duration and severity of the attacks increasing with time. A definite relation to the ingestion of fatty or greasy foods was elicited. There was no history of icterus, clay-colored stools, hematemesis, melena, constipation or diarrhea. The attacks were not severe enough to require hospitalization.

Except for mild dyspnea on exertion, the remaining past history was not significant.

Physical Examination: Temperature 100° F. pulse 72; respirations 15; blood pressure 116/70. Positive findings included slightly icteric sclerae, marked tenderness in the right upper quadrant beneath the costal margin, voluntary rigidity and a positive Murphy sign. No abnormal abdominal masses were felt. Otherwise, the examination revealed nothing unusual.

Laboratory Data: A blood count revealed the hemoglobin to be 14 grams; RBC 4.87 million, WBC 9500 with a differential of 68 per cent polymorphonuclears, 19 per cent stabs, 1 per cent metamyelocytes, 8 per cent lymphocytes, 3 per cent monocytes and 1 per cent eosinophils. Icterus index, 19 units. Urinalysis revealed the presence of bile and a trace of albumin.

Hospital Course: On admission, treatment consisted of intravenous fluid, a fat-free diet, vitamin K, and morphine as needed. Since the clinical condition was becoming worse, the Senior Surgeon advised immediate operation, which was performed forty-eight hours after admission.

Operative Report: Under sodium pentothal induction and cyclopropane anesthesia, an upper right rectus incision was made. Upon opening the peritoneal cavity, approximately 700 cc. of free bile were found. Careful examination disclosed four facetted yellow calculi, each 4 mm. in diameter, in Morrison's pouch. The gallbladder was markedly injected and distended; plaques of exudate were noted. Gentle palpation of the common duct produced three other calculi of similar nature. However, the surgeon, at the time, felt that the calculi had escaped from a necrotic zone in the neck of the gallbladder. The latter was removed. At this time, the patient's condition was extremely poor; therefore, a split hard rubber drain was placed in the common duct and a Penrose drain in Morrison's pouch. The abdominal wound was closed in the usual manner.

Pathological Report: Acute purulent exacerbation of a chronic cholecystitis, no evidence of perforation. Thirty facetted calculi (3 to 10 mm. in diameter) of mixed pigment type.

Postoperative Course: The postoperative course was exceedingly stormy. The patient received intravenous fluids, including amigen, blood and plasma; the usual supportive measures were given. In addition, penicillin and streptomycin were administered. However, the patient's condition grew steadily worse. On November 10, 1948, the lower portion of the abdominal wound was opened, allowing a large amount of bile to be evacuated. The patient's condition was such that further operative procedures were inadvisable. The patient expired ten hours later.

Postmortem Examination: The significant findings may be presented as follows:

The peritoneal cavity contained approximately 1000 cc. of yellow bile-colored fluid. The parietal and visceral peritoneal layers contained a yellow fibrinous-like exudate. The intestinal loops were moderately edematous and bound together by fresh fibrinous adhesions which were easily broken. The liver extended 4 cm. below the costal margin in the midclavicular line. Its surfaces were covered by a shaggy bile-stained exudate. The liver itself, weighed 1390 grams. The hepatic parenchyma was reddish brown in color, friable in consistency and the locular pattern was faintly visible. There was a free ooze of blood from the cut surfaces. The intrahepatic ducts were slightly dilated and patent.

Examination of the gallbladder bed revealed a Gel-foam pack in this area. The cystic duct had been doubly ligated approximately 1 cm. from the common bile duct. Examination of the common and major hepatic ducts revealed considerable dilatation. The common bile duct varied from 1 to 1.3 cm. in its luminal diameter. All extrahepatic ducts with the portal vein and hepatic artery were embedded in markedly edematous bile-stained connective tissue and fat. Careful dissection of the common duct revealed a 3 mm. perforation on the posterior aspect approximately 4 cm. above the

Figure 1

Figure 2

Figure 3

ampulla of Vater (Fig. 1). A small sinus extended upward nearly to the origin of the cystic duct and this passage contained three facetted yellow calculi each measuring 3 mm. in diameter. Upon gently milking the common duct, three similar calculi were extruded through the perforation into the above described sinus. In addition, there was a free passage of bile. Then the common duct was opened longitudinally revealing the area of perforation (Fig. 2). The mucosa, itself, was smooth and yellowish brown in color and no obvious exudate was present. However, at the site of perforation, the edge of the mucosa was rough and irregular. Further inspection revealed an 8 mm. greenish black smooth calculus, firmly embedded in the ampulla of Vater (Fig. 3). Considerable difficulty was encountered in its removal at the postmortem table. The portion protruding into the duodenum was brownish black in color while the portion within the duct was greenish brown in color. No significant abnormalities were encountered in the major hepatic ducts.

Histopathologic study of the common bile duct disclosed a subacute inflammatory process in which the greater portion of the mucosa was intact. At the site of perforation, however, there was necrosis with a rather intense inflammatory reaction and complete disintegration of the wall (Fig. 4 and 5). Apparently, the rupture was due to pressure necrosis by a calculus and increased intraductal pressure. Study confirmed the presence of bile peritonitis and septic peritonitis due to *Escherichia*

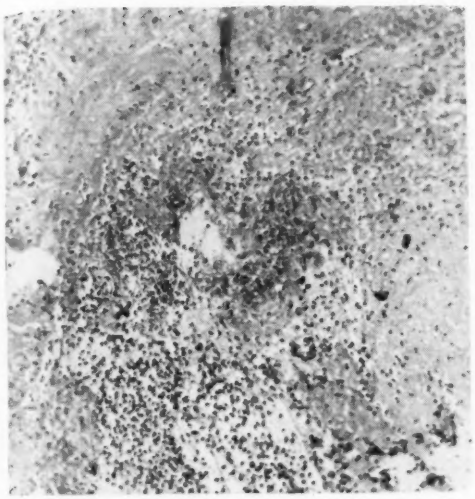

Figure 4

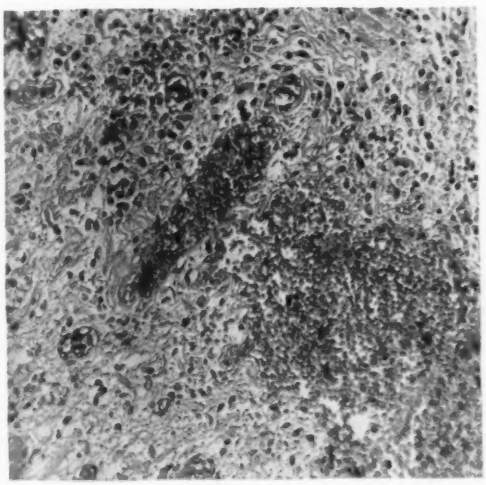

Figure 5

coli. The liver disclosed some middle and central zonal necrosis associated with bile stasis. Moderate parenchymatous degeneration of the myocardium and changes of terminal vascular failure were noted.

Comment: Clinically, such a case represents an acute abdominal emergency requiring closure of the perforation and adequate peritoneal drainage, supplemented by good supportive measures. In this case, there was a forty-eight hour observation period prior to surgical intervention. Further, the opening in the common bile duct was in a posterior position and not recognized. A fatal outcome ensued.

Case No. 2. White female, age 48, admitted September 30, 1949; discharged October 20, 1949.

Chief Complaint: Right upper quadrant pain.

Present Illness: The patient had been in excellent health until September 27, 1949. At 10:00 P. M. she had eaten a meal of chop suey and some twenty minutes later experienced epigastric distress associated with frequent belching. The following day, right upper quadrant pain developed which radiated to the right and infrascapular area. The pain was colicky in type and became progressively severe. There was some nausea and vomiting. No jaundice was noted. No previous similar episodes or history of gallbladder disease was elicited.

Physical Examination: Physical examination disclosed generalized abdominal tenderness, rigidity, rebound tenderness and a positive Murphy sign. The Senior Surgeon's impression was that of acute cholecystitis with possible rupture of the gallbladder. Appendicitis with perforation and peritonitis was considered.

Laboratory Data: The significant laboratory data on admission may be outlined as follows: RBC 4.20; Hgb. 12.9 grams; WBC 15,400 with a differential of 75 per cent polymorphonuclears, 10 per cent stabs, 12 per cent lymphocytes, 3 per cent monocytes and 1 per cent eosinophils. Toxic granulation was evident.

The NPN was 28 mgms; plasma chlorides 570 mgms; carbon dioxide combining power 58 vol. per cent; total serum proteins 6.8 grams with normal AG ratio.

Except for a trace of albumin, no abnormalities were found in the urine.

Course in the Hospital: The Senior Surgeon felt that immediate operation was indicated. Under general anesthesia of sodium pentothal supplemented with nitrous oxide, a right rectus incision was made in the upper quadrant. Upon incision of the peritoneum, approximately 1000 cc. of the muddy dark grey bilelike fluid was encountered. No food particles were seen in this fluid. No areas of perforation were found in the duodenum or stomach. The gallbladder was hyperemic and markedly edematous; its wall was greatly thickened. The common duct was identified in a greenish fibrinous exudate. Careful examination disclosed a 3 to 4 mm. perforation located in the anteromedial aspect 1 cm. distal to the junction with the cystic duct. The gallbladder was aspirated but bile could not be obtained. The fundus was incised and four firm, greenish black calculi were removed. These stones were of pigment type with some calcium salts; they varied from 2 to 5 mm. in diameter; the external outlines varied from round-oval-faceetted. The common duct was explored and a probe entered the duodenum easily. Clear bile escaped from the perforated area in the common duct. The zone of perforation was enlarged and a "T" tube was in-

serted. A hard rubber drain was placed in the fundus of the gallbladder and a Penrose drain was tucked in the foramen of Winslow. The drains and "T" tube were brought out through a small incision lateral to the abdominal operative site. The peritoneum was closed with continuous plain catgut sutures. The anterior sheath of the rectus muscle was closed with interrupted figure-of-eight chromic catgut sutures. Subcutaneous tissues were approximated with plain catgut sutures and the skin closed with interrupted vertical mattress silk sutures. The patient returned to her room in good condition.

During the operation the patient received 1000 cc. of 5 per cent dextrose in saline.

Postoperative Course: The immediate postoperative course was fairly good. Wagensteen drainage was used. On November 2, 1949, the temperature was still 103°F. The abdomen was slightly distended and generally tender. We thought that we were dealing with a bile peritonitis, with or without secondary bacterial invasion. Therefore, the patient was given 300,000 units of penicillin per day and 250 mgm. of streptomycin every three hours for the next eight days. Supportive intravenous fluids, including blood, plasma, Baxter's protein hydrolysate, and vitamins, were employed. During this period, the temperature and pulse gradually returned to normal. The fluid and electrolytic balance was carefully followed by laboratory procedures. The drains were removed on November 8, 1949.

Figure 6

On November 11, 1949, a cholangiogram (Fig. 6) failed to reveal calculi and the dye passed easily into the duodenum and cystic ducts. The entire biliary tree was well outlined. The patient's condition steadily improved and she was discharged on her twentieth hospital day in excellent condition.

Comment: This case illustrates the results of prompt surgical exploration, evacuation of free bile, closure of perforation in the bile duct, good peritoneal drainage and adequate supportive measures. Plasma, whole blood, and antibiotics must be used. Unless this regimen is followed, death may be expected in nearly all cases.

DISCUSSION

Choledochal calculi are relatively frequent and represent the most important factor in perforation of the common bile duct. The source of these stones is somewhat controversial though their their nature and character, except for size, are identical with those of calculi found within the gallbladder. Whether they arise entirely within the duct, or represent deposits of crystals about a nidus expelled from the cystic duct, matters little to this discussion. The symptoms, signs, and laboratory findings are essentially those of cholelithiasis with the added factors incident to varying degrees of obstruction to the bile flow. The treatment of the latter is fairly uniform, i.e., immediate relief of the obstruction by removal of the calculus or calculi. There have been instances where failure to remove all calculi has resulted in postoperative ruptures in the biliary tract.

It is generally agreed by most writers, particularly Newburger, Brunshwig, Newell and McWilliams, that the following factors are important in the perforation of the bile duct system.

1. Increased intraductal pressure due to mechanical blockage by calculi, mucus plugs, tumors, parasites, and stricture, or by reflex spasm of the sphincter of Oddi, or both. Naturally, overdistention will result in perforation at the weakest point.

2. Active cholangitis with necrosis of the ductal wall.

3. Thrombosis of arterial or venous channels may lead to ischemic necrosis and perforation. Calculi may or may not be associated.

4. Activation of refluxed pancreatic juice into the common bile duct may produce tissue necrosis. This supposition seems unlikely in view of our present day knowledge.

Trauma to the duct from external sources, such as falls and automobile accidents, direct wounds caused by knives and projectiles, upward traction due to shrinkage of the liver or downward traction by adhesions, and carcinomatous invasion of the duct (carcinoma of the stomach or pancreas) are considered remote possibilities.

Rupture, traumatic or otherwise, of any portion of he ductal system or gallbladder leads to bile peritonitis. McLaughlin,[23] lists a very complete and satisfactory classification of the possible factors in the etiology of bile peritonitis. Obviously, one of the causes is rupture of the common bile duct. His conclusion that the vast majority are due to microscopic rather than gross rupture may be questioned. The role of calculi cannot be underestimated.

As in gallbladder disease, the highest incidence is in the fifth decade of life or later. It is more common in the female than in the male, and there is an antecedent history highly suggestive of cholelithiasis.

Clinically, the condition is characterized by rapid onset, severe pain, often some degree of shock, frequently subclinical jaundice and obvious signs of peritonitis. Including those incident to recent surgery, either cholecystectomy with perforations of the cystic duct at its stump or a rupture of a sutured common or hepatic duct, the histories suggest gallbladder disease of a severe degree. Differentially, acute pancreatitis or a perforated peptic ulcer must be seriously considered. At operation, free bile in fairly large amounts is found within the peritoneal cavity.

The disease is associated with a mortality of 50 to 70 per cent in most recorded series. It seems likely that the shocklike picture is, in part at least, due to a large fluid loss and marked electrolytic changes in the body. The use of antibiotics may be indicated since pathogenic organisms belong to the *Escherichia coli group*, although hemolytic streptococci may be encountered.

SUMMARY

Two additional cases of spontaneous, nonoperative rupture of the common bile duct have been presented with a general review of the literature. Prompt recognition of this rare clinical entity is mandatory since operative intervention is imperative in these cases. Where there is gross rupture of the common bile duct, the mortality is nearly 100 per cent if operative correction is not performed. Likewise, postoperative supportive therapy with proper laboratory aids cannot be overemphasized.

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URETEROSIGMOID ANASTOMOSIS AND CYSTECTOMY*

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For many years, the operation of ureterointestinal anastomosis and cystectomy carried such a formidable operative mortality and morbidity that it was given little attention in the role of treatment for carcinoma of the bladder. However, with the advent of antibiotics and improved surgical technics, this procedure is not nearly as formidable as was formerly believed, and is rapidly acquiring a wide acceptance in the treatment of many conditions requiring diversion of the urinary stream.

In this presentation the following points will be presented: (1) Historical background; (2) Operative technic; (3) Postoperative results and complications; (4) Indications for cystectomy and ureterosigmoid anastomosis.

HISTORICAL BACKGROUND

In this discussion I have taken the liberty of drawing freely on the excellent article written by Hinman and Weyrauch in 1936,[1] who summarized all of the methods used up to that time. As early as 1879, T. Smith reported the first ureterointestinal anastomosis. Interestingly enough, he utilized a direct end-to-side anastomosis, very similar in nature to the one we will describe. However, due to technical difficulties the operation was unsuccessful. In 1892, Chaput described a similar method which did not appear to be too successful. However, this fact did not seem to alter his enthusiasm, for he states: "It is particularly indicated in complete resection of the bladder, cancer, tuberculosis, extrophy, in fistulae of the ureter, and in wounds and ruptures of that organ, and in certain cases of ureteral calculus." I do not believe that in light of our present knowledge we are quite as enthusiastic about the indication as was Chaput.

Through the years there followed a long series of various methods and devices for this procedure. Their very number is staggering and indicative of the fact that none was too successful. No attempt will be made to cover all of them. However, there are a few which are of sufficient significance in principle to warrant mention. The one principle which seemed to have some merit and for which I believe there is a definite clinical indication at present was the implantation of the ureters to an excluded portion of the intestinal tract. Of these, the one which seems to be the most practical is an artificial bladder made from the entire rectum with an iliac sigmoidostomy, first described by Mouclare in 1895, and later recommended by Miles, to avoid ascending infection.

In 1909, Coffey elaborated the submucosal, or tunnel technic. He later modified this method to a certain extent, by the use of ureteral catheters (Coffey #2 technic) and then had a Coffey #3 technic using a necrotizing suture, which had very little use and was promptly abandoned. The Coffey #1 technic and the many modifications thereof, had almost complete acceptance by the entire urologic profession for many years. This method is still widely utilized and, no doubt, produces excellent results in many instances. However, it is my belief that the method is faulty in principle. It is believed by many that the submucosal tunnel per se, does not produce a true valve action. In addition, constriction by surrounding tissue and scar tissue laid down postoperatively is apt to produce partial obstruction at the stoma with a consequent hydroureter and hydronephrosis, resulting in urinary sepsis and the loss of kidney function. The belief that the portion of

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*Presented at the Seventy-first Annual Meeting of the Louisiana State Medical Society, New Orleans, La., May 9, 1951.

ureter drawn into the bowel sloughs away to produce a smooth stoma, is completely false. As a matter of fact, this portion of the ureter very frequently persists and forms a source of obstruction.

In 1948, Nesbit[2] and the writer simultaneously and independently, arrived at the conclusion that the elimination of the submucosal tunnel, with the production of a good stoma would produce a better end result than the previous methods described. Although our methods are similar in the principle that a direct mucosa to mucosa anastomosis is established and the submucosal tunnel is eliminated, they vary considerably in technical details. Subsequently, Frank Hinman Jr.[3] presented a method of implantation very similar in principle to the one to be presented.

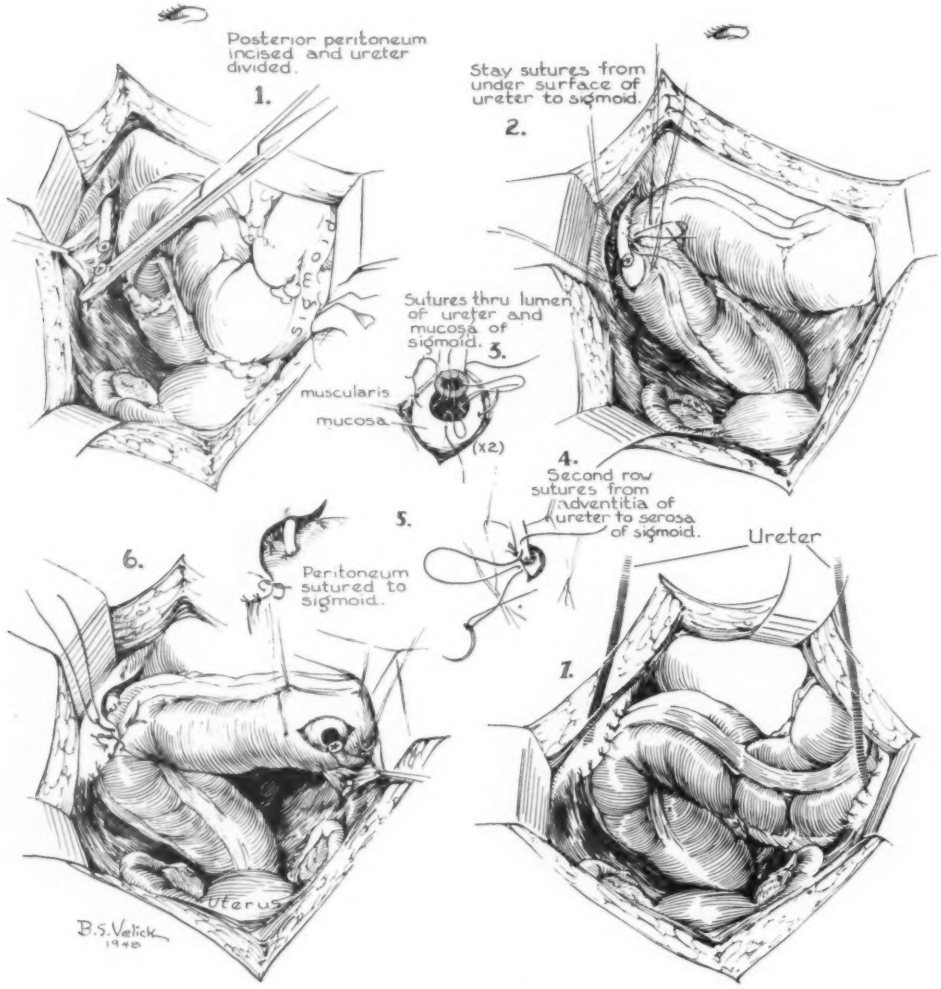
PREOPERATIVE AND POSTOPERATIVE CARE

Preoperative care is the same as for any intestinal surgery. The patient is prepared for five days on a low residue diet and given sulfathalidine in the usual doses. In patients who are sensitive to the sulfonamides, streptomycin may be used in doses of 4 gm. by mouth daily for two days and a quite satisfactory preparation obtained. Cleansing enemas are given the night before surgery. Postoperatively, the patient is maintained on a nothing by mouth regime. Adequate fluid intake is given intravenously. The patient is then placed on a low residue diet, and is usually on a regular diet by the fifth postoperative day. A colon tube is maintained in the rectum for four to five days. Both penicillin and streptomycin are given for the first seven postoperative days.

OPERATIVE TECHNIC

I should like to emphasize at the outset of this discussion the importance of meticulous attention to detail in the placing of sutures, the preservation of blood supply, and the gentle handling of tissues. This is truly a plastic procedure, and if it is to be successful, the strictest attention to all minute details is essential.

With the patient in the extreme Trendelenburg position, the abdomen is opened through the usual midline subumbilical incision, usually extended up above the level of the umbilicus. After the peritoneum is opened the small intestine is packed out of the pelvis, and the pelvic and aortic areas carefully palpated for the presence of lymph node involvement. The sigmoid is then manipulated to determine the best site of anastomosis. The site chosen is the point at which the sigmoid lies most naturally over the course of the ureter. It is of fundamental importance that the bowel be brought to the ureter, rather than the ureter to the bowel. The right side is usually anastomosed first. After the site of anastomosis has been determined an incision is made into the posterior parietal peritoneum directly over the ureter. The ureter is then located and isolated for a distance of about 3 cm., care being taken to preserve the blood supply. It is then cut across and the lower segment ligated. The medial leaf of the incision in the posterior parietal peritoneum is then sutured to the lateral wall of the sigmoid using silk sutures. This serves a twofold purpose, in that it "fixes" the site of anastomosis and forms the under surface of the peritonealizing closure to be made later. When this step is completed, a longitudinal incision is made through the serosa and muscularis down to the submucosa of the sigmoid. It seems to make little difference whether this incision is made through a tinea or not. When the submucosa has been reached it protrudes into the incision very much as noted in Figure 2. The ureter is then brought over to the site of the anastomosis as shown in Figure 2, care being taken to avoid angulation or kinking of the ureter. The first suture is a fixation stitch, which simply fixes the ureter above the site of anastomosis. A second suture is then taken between the upper angle of the incision down through the muscularis of the bowel and the adventitia of the ureter. Both of these sutures are so placed that the end of the ureter will be directly in the center of the incision into the wall of the bowel. A stab opening is then made through the mucosa and submucosa. This opening should be ap-



Figures 1-7

proximately the same size as the end of the ureter. A direct mucosa to mucosa approximation is then obtained, the end of the ureter and the submucosa of the bowel being approximated as shown in Figure 3. Four O chromic catgut is used for this purpose. The sutures are so placed that the knots are on the outside, care being taken to avoid occlusion of the lumen of the ureter or puckering at the site of anastomosis. A second layer of sutures is then placed between the adventitia of the ureter the muscularis and serosa of the bowel as shown in Figure 4. These sutures should be taken in the longitudinal axis of the ureter to avoid disturbance of blood supply at the distal end. They are placed just far enough up on the ureter to produce a slight invagination of the first suture line. This creates a "papilla" within the lumen of the bowel and may be of some value in the prevention of the reflux of intestinal contents. When the second layer of sutures is completed, a good water tight junction without stenosis should result. You will note that an accurate approximation of mucosa to mucosa is present. It is a fundamental surgical principle that in order to avoid scarring at the site of anastomosis in any type of bowel surgery this approximation must be ob-

tained. After the anastomosis is completed the lateral leaf of the incision in the posterior parietal peritoneum is elevated, care being taken that no kink is produced where the ureter comes out from beneath it at the upper angle. The peritoneum is then sutured to the serosa of the sigmoid as shown in Figures 5 and 6, thereby completely extraperitonealizing the anastomosis. The same procedure is repeated on the left side as shown in Figures 6 and 7. More recently, we have been placing the ureter at a lower level in the sigmoid, in the belief that there is probably less absorption of electrolytes with the ureter placed at a lower level. This adds no particular technical difficulty, except that one is working somewhat deeper in the pelvis than with the original method.

IMMEDIATE AND LATE MORTALITY AND MORBIDITY

A total of 64 successive cases have been done by this method with two postoperative deaths, all other patients having been discharged from the hospital under their own power. Many of these were exceedingly poor operative risks, one of the two operative fatalities being a markedly debilitated 70 year old male, and I believe we exercised poor judgment in doing too much surgery at one sitting.

The average age was 61, the youngest operated for malignant disease being 38, and the oldest 80. Fifty-one patients had bilateral ureterosigmoid anastomosis and cystectomy in one stage. Six had two stage procedures, and in 7 cases, ureterosigmoid anastomosis alone was done. It is our belief that the cystectomy is much easier to perform at the time of ureterosigmoid anastomosis, and that the added surgery does not increase the operative risk any more than a second stage cystectomy would do.

Immediate postoperative complications have been relatively uncommon. There were 4 postoperative eviscerations and 3 incisional hernias. We believe these were in part due to the modified Cherney incision which was used on all the earlier cases. Since changing to a midline incision, this complication has not occurred. Two cases had troublesome ileus for several days. There was 1 retropubic abscess and 1 case of acidosis. Three patients developed fecal fistulae. However, each of these was due to rectal involvement by carcinoma or surgical trauma and had no apparent connection with the anastomosis.

The late results are shown in Table 1:

TABLE 1.

Years	Alive	Dead
0—1	1	21
1—2	13	9
2—3½	9	1
Total	29	31

Follow up on 4 cases incomplete.

Of the total of 60 patients, 29 or 48 per cent are still living. Although 52 per cent of the patients operated upon are dead, we wish to emphasize that no case has been refused surgery which we felt could stand the operative procedure. A great many cases with advanced carcinoma, in whom little or no hope for cure was held, have been operated upon with the belief that the palliation obtained was worth the surgery involved. In our experience this belief has been well substantiated. Of the 21 cases dying in the first year, 16 died of carcinoma, 2 deaths were renal and 1 was due to a pelvic abscess. The other 2 died of unknown causes. One patient who expired after twenty months died of acidosis, which was recognized too late, and improperly treated. At autopsy, there was no evidence of recurrence of his carcinoma. Therefore, only 8 per cent of the total deaths may be directly attributed to the ureterosigmoid anastomosis. Admittedly, the results of our cancer surgery leave much to be desired.

URINARY SEPSIS

Since all patients were placed on antibiotics during the immediate postoperative period early sepsis presented a problem in only 1 case and disappeared after about two days of rectal drainage. None of the other cases presented any evidence of urinary sepsis in the immediate postoperative period.

Of a total of 60 cases in which an accurate follow-up was obtained, 44 or 60 per cent have had no chills, fever or other evi-

dence of urinary sepsis. Fourteen had a minimal amount of chills and fever. In this category we considered those who had had two or less episodes of sepsis. Eight were considered to have had moderate urinary sepsis and, two are listed as severe. In one of the latter, kidney infection was undoubtedly the primary cause of death, although acidosis may have played a part. In the other, the patient was a severe hypertensive preoperatively, and had an aneurysm of the abdominal aorta. However, he did experience considerable urinary sepsis and, therefore, it was felt that in all fairness we must consider it as a contributory factor in his demise.

In general, I believe that we can state that urinary sepsis has not presented any great problem in these cases. Each episode of infection has, with one exception, been readily controlled by the usual antibiotics or chemotherapy, and has been of relatively short duration. In addition, we have observed that as the patient continues postoperatively, there is apparently some degree of natural immunity built up to the intestinal organisms. In several patients, we have observed a decreasing severity both in the character and frequency of the attacks.

AIR PYELOGRAMS AND URETERAL REFLUX

The presence of air pyelograms following direct anastomosis has been mentioned as an objection to this method. In our experience, this has not been the case. All postoperative pyelograms up to as long as two years postoperatively were reviewed for air pyelograms. Air was found to be present in the kidney pelvis in only five kidneys from the entire series. With one exception, these were present in kidneys which were nonfunctioning preoperatively. It is interesting to note that these patients had very few symptoms and sepsis was not a pronounced problem in any of them. If one transplants a hydronephrotic ureter, with marked fibrosis in the musculature, and poor peristalsis, it is inevitable that postoperative difficulties will develop.

A total of 16 patients was studied for reflux, the study being made by filling the colon with a Skiodan enema under considerable pressure, and then shooting a flat film. This study included only 31 kidneys, 1 patient having had a nephrectomy prior to ureterosigmoid anastomosis. Of the 31 kidneys, 5 showed a reflux of Skiodan up the ureters. In only 1 patient was the reflux bilateral.

EARLY AND LATE INTRAVENOUS PYELOGRAPHY

The total number of patients with accurate pyelograms and follow-ups was 46. For the purposes of more accurate evaluation, the data is broken down into the total number of kidneys. A preoperative and postoperative comparison is incorporated in Table 2.

TABLE 2.

Total No. of Patients With Accurate Pyelograms and Follow-up—46

	Preoperatively	Postoperatively
No. kidneys Studied	92	91
Nonfunctioning	9	7
Hydronephrosis	18	31
Normal	62	41
Decreased function	3	12

A number of the postoperative hydronephroses so listed were minimal in character. Many patients who showed a mild dilatation immediately postoperative, had a definite reduction in dilatation on films taken at a later date. It is noteworthy that 2 kidneys which were listed an non-functioning to intravenous pyelography preoperatively regained some function postoperatively. However, the number of normal kidneys was reduced from 62 to 41, or a decrease of approximately 34 per cent. Likewise, decreased function was noted in 12 kidneys. In most instances these showed poor dye shadow, but as far as could be determined, no actual hydronephrosis existed. Although many factors contribute to faint shadows in intravenous urography, we consider these results as unfavorable. Repeated pyelograms were made on many of these patients, some as long as three years postoperatively. In only 1 case did we note progression of the hydronephrosis. Surprisingly enough, this patient has no urinary sepsis and is maintaining a normal nonprotein nitrogen.

ACIDOSIS AND HYPERCHLOREMIA

One of the major problems in caring for the patient with ureterosigmoid anastomosis is that of acidosis. Very early in our experience we encountered an extremely interesting case, which emphasized this problem, but it was not until subsequent cases of acidosis began to appear that we realized its true significance. This first patient was a 54 year old colored man, who was admitted to the Veteran's Hospital with extensive carcinoma of the bladder, and involvement of both ureteral orifices to such an extent that intravenous pyelography revealed a completely nonfunctioning kidney on the left, and only slight function with marked hydronephrosis on the right. Urea nitrogen on admission was 42, and he appeared to be in a very serious condition. It was concluded that his surgery should be done in two stages. The ureter of the functioning kidney was transplanted to the sigmoid, and ten days later the second ureter was transplanted and the cystectomy done. The patient had a fairly stormy postoperative course but made a comparatively good recovery, only to return to the hospital about three weeks after discharge with an N.P.N. of 155 and a CO_2 of 12. He was comatose at that time, and required a great deal of intravenous molar lactate to correct his chemistries. However, he responded surprisingly well to treatment and was discharged on sodium bicarbonate by mouth. Since that time, he had several hospital readmissions, and each time he was found to have a moderately severe acidosis. However, his general condition remained good and he continued to work at his occupation of cleaning out boxcars for a cement company some two years postoperatively. He finally expired after twenty-seven months of acidosis. This patient stimulated us to make further studies on those patients with ureterosigmoid anastomosis, and we have endeavored to follow as many as possible from this standpoint.

Clinical symptoms which we have encountered have been general malaise, poor appetite, mild nausea, and weakness.

Accurate chemical studies have been obtained on 21 patients. Sodium and potassium levels were normal in the 13 cases in which these determinations were made. Practically all patients showed a moderate elevation of blood chlorides whether acidosis or elevated blood nonprotein nitrogen was present or not. Of the 21 cases studied, 33 per cent had CO_2 values of 50 vol. per cent or above. These were considered to be normal. Twenty-four per cent had a CO_2 level of 40 to 48 vol. per cent, and 43 per cent had CO_2 levels below 40 vol. per cent. All but two of these were between 30 to 40 vol. per cent. It is noteworthy that an elevation of blood nonprotein nitrogen occurred in only those patients who had advanced kidney damage and were listed as having expired from kidney disease. In none of the others was there any appreciable elevation of the nonprotein nitrogen, either early or late. There seemed to be little relation between the CO_2 level and the blood nonprotein nitrogen. This fact emphasized the point that a simple determination of nonprotein nitrogen is not sufficient to evaluate the condition of a patient who has had a ureterosigmoid anastomosis. We believe that our incidence of acidosis is no higher than that reported by other methods.

Eleven of these patients were placed on rectal drainage for a five day period, but this seemed to have no appreciable effect on the CO_2 levels.

Boyce,[4] in a recent study of the absorption of certain constitutents of the urine from the large bowel of dogs, was of the opinion that the low levels of serum carbon dioxide combining power were due to the loss of base both from the diarrhea induced by the flushing of the colon with urine, and by inefficient loss of base from a kidney functioning at maximum excretory capacity. In addition, he was of the opinion that the retention of acid radicals might be a contributing factor. It is noteworthy that his experimental animals showed an initial depression of carbon dioxide combining power, which later returned to a constant subnormal level. This has certainly not been our clinical experience. In only one

or two of our patients was there any evidence of lowered CO_2 capacity immediately postoperatively, and in most instances this condition appeared after a period of two or three months. Ferris and Odel,[5] in analyzing the electrolyte pattern of the blood in some 141 patients, found acidosis in 88 per cent, and hyperchloremia in 80 per cent. It was their belief that the hyperchloremic acidosis seemed to result from the absorption of chloride across the rectal mucosa and that the acidosis was directly secondary to the hyperchloremia. It is my opinion that a combination of two factors is present. First, with the hyperchloremia an excess of sodium is bound up as sodium chloride leaving less available as bicarbonate. Second, with any ureterosigmoid anastomosis there is a certain inevitable degree of kidney damage. There is a wide individual variation both in the amount of damage which is already present preoperatively, and that which occurs postoperatively. If the kidneys are normal or nearly so they are able to compensate for hyperchloremic acidosis by an increased excretion of fixed acids. However, with kidney damage this compensation cannot take place and clinical acidosis occurs. This explanation would account for the fact that a certain percentage of these patients does not show any laboratory evidence of a lowered CO_2.

As regards management of this rather serious complication, I believe that there are several factors to be considered. First, one must be aware that this complication is a very common one, and these patients should have blood studies at regular intervals, with blood chloride and CO_2 levels, as well as nonprotein nitrogen determination. The awareness that this complication does exist will at least preclude some of the unfortunate experiences which we have had prior to our recognition of it. As far as instructions to the patient are concerned, we usually apply the following regime: (1) The patient is instructed to use only the amount of salt in his diet required for cooking, and to add no additional salt to his food. It is our belief that a salt free diet is impractical because we feel that these patients need adequate nutrition and since a completely salt free diet is rather unpalatable, they should not be restricted too rigidly. (2) The patient is instructed to empty the bowel at two hour intervals during the day, and as often at night as he awakens. (3) If any tendency to acidosis exists, the patients are given 4 grams of sodium bicarbonate three times daily. This seems to control the acidosis quite satisfactorily in practically all instances. Sodium citrate and one sixth molar sodium lactate by mouth have also been used for this purpose instead of sodium bicarbonate.

Recently, Bricker,[6] of Washington University, in doing pelvic sweeps for extensive carcinoma of the pelvic organs has devised a bladder consisting of a short loop of ileum terminating in an ileostomy. This consists of a very short loop from which absorption is minimal, and the urine does not collect in it, but flows continuously into a Rutzen bag, through a wet ileostomy. The method of transplantation of the ureters into the loop is that which we have described. He reports a complete absence of sepsis and acidosis in his series of 25 cases. About six months ago, we established a rectal bladder, performing the transplants in the usual fashion, and following this procedure with an iliac colostomy, closing off the distal end of the rectosigmoid to form a bladder. This patient has had an excellent course from a standpoint of both sepsis and acidosis. However, he is of a fairly low intellectual level and complains rather bitterly of his colostomy. Although Bricker's results seem to be excellent, it is my belief that a wet colostomy is highly undesirable and should be avoided if an intact sigmoid colon is present. The formation of an artifical bladder from the sigmoid and rectum is certainly practicable, and in my opinion should be used in all cases where a preexisting hydronephrosis and hydroureter exist. We have noted that in almost all of the cases in which we have had trouble, our difficulties have arisen from attempting to transplant large ureters into the bowel. These are the cases which developed air

pyelograms and sepsis. However, this is not entirely true, as a few of our cases of sepsis have had essentially normal pyelograms. As for the routine employment of the artifical bladder, the problem seems to be whether the disadvantages of a permanent colostomy, with an inevitable increase in operative mortality due to added surgery, are great enough to overbalance the degree of sepsis and acidosis present in a certain percentage of the cases. We are at present attempting to build up a series of cases with colostomies, so that we may evaluate this problem more accurately.

INDICATION FOR CYSTECTOMY AND URETEROSIGMOID ANASTOMOSIS

Perhaps the most controversial subject in the entire field of urology is the question of when to do a cystectomy for carcinoma of the bladder. Opinions among top flight urologists vary from those who state that cystectomy is never indicated, to those that feel that it should be performed in almost all cases of grade two, or above, papillary carcinomas of the bladder. The first group contends that grade three and four tumors are incurable by any method, and that if the grade two tumors are curable surgically, they can also be cured by transurethral means or radiation. The second group feels that the grade two tumors are very apt to grow and progress, and that if a cure is to be obtained, early removal of the entire carcinoma before any spread has occurred offers the best prognosis. Undoubtedly a middle path somewhere between these two extremes offers the soundest approach to the problem.

In my opinion, partial cystectomy, although recommended by some excellent men such as Jewett,[7] is fundamentally unsound surgically. The lymphatic networks of the bladder originate in the mucous and muscular coats, being particularly abundant in the muscular coat, whence they drain into the superficial lymphatic plexuses which pass to the surface of the bladder and there give rise to the collecting trunks. It is readily apparent that with any muscle invasion whatsoever, the possibility of lymphatic extension of carcinoma beyond the area of excision would be great. I believe that it is no more logical to do a partial cystectomy than it is to do a partial mammectomy for carcinoma of the breast. Admittedly, one will occasionally cure a lesion by this method, but the percentage of cure would be so small as to preclude its usefulness.

The use of Broder's classification alone in determining the indications for cystectomy is not satisfactory. It has been repeatedly pointed out that different grades of malignancy are present in the same tumor and that the specimen obtained at the time of cystectomy may show considerably more malignant change than was anticipated from the obtained biopsies. Therefore, one must use other criteria as well as the grade of malignancy.

Immediate cystectomy should be performed on all grade three and four tumors and tumors of the sessile or invasive type, irrespective of size. The smaller the tumor, the more radical the procedure. The use of bimanual recto—or vagino—abdominal palpation under anesthesia, as suggested by Jewett, is very worth-while in determining operability. However, I do not feel that any patient should be denied the palliation obtained by diversion of the urinary stream, even though the bladder carcinoma has advanced beyond the stage of operability. I am sure we have all had occasion to observe patients dying of carcinoma of the bladder, and are familiar with the inevitable blood clots, sepsis, continuous desire to void, and other unhappy complications from which these poor people suffer. On the other hand, since we have been performing ureterosigmoid anastomosis even in inoperable cases, we have been particularly impressed with the comparative comfort which these people have. I am sure we have prolonged their lives considerably in many instances, and made their last days far more comfortable than they would have been without diversion of the urinary stream. Admittedly, the chances of cure in grade three and four invasive lesions are poor. However, the prognosis by any other method is completely hopeless. It has been suggested

that the manipulation involved in cystectomy hastens the spread of carcinoma, and shortens the life of these patients. I am somewhat doubtful that this is the case, but even if it does we have at least given them a moderate degree of comfort in their last days.

The grade two papillary lesions with a fairly wide base are the ones which have caused me the most difficulty in reaching a decision as to what procedure to follow. Several factors should be considered, in making up one's mind in these cases: First, the gross appearance of the tumor, its location, whether multiple or single, and the width of the base. The obtaining of an accurate biopsy from the base of the tumor in these cases is unquestionably the most important single factor in determining whether to operate or rely on transurethral resection of the tumor. It has been my practice on these patients to do as complete a transurethral resection as possible separating the tissue removed from the base of the tumor from the more superficial tumor tissue, so that an accurate determination can be made as regards muscle invasion. If there is definite muscle invasion then it is my belief that cystectomy should be performed immediately upon making the diagnosis. If, on the other hand, there appears to be no muscle invasion, these patients may be observed cystoscopically over a period of time. If the lesion is at all extensive at the time of the first transurethral, the patient is returned at the end of a two month period and even though the mucosa appears healed at the site of the lesion, additional tissue is removed with a resectoscope to determine the presence or absence of carcinoma cells beneath the mucosa. I am sure that we have all had the experience of having removed a surface carcinoma and having had extension beneath the healed mucosa with the tumor appearing in an adjacent area in a considerably more extensive form than the original lesion. It is to preclude the loss of valuable time that this second biopsy should be done. If no carcinoma cells are found at this time the patient may be observed at regular intervals and cystectomy deferred.

Time does not permit the discussion of the relative merits of radiation therapy or electrocoagulation.

CONCLUSION

I believe that we have developed a practical method for ureterosigmoid anastomosis. This belief is borne out by the fact that all of the patients with nonmalignant disease, and some with malignant disease, are alive and in comparatively good health well over three years after transplantation. The results of our cancer surgery leave much to be desired. The defeatist attitude which has been assumed by some concerning carcinoma of the bladder should not be adopted. We should continually endeavor to improve our cancer surgery, and in this way more satisfactory results may be obtained.

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THE VALUE OF ELECTROENCEPHALOGRAPHIC EXAMINATION*

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NEW ORLEANS

The electroencephalogram is an adjunctive, diagnostic instrument. It is to the brain effectively what the electrocardiogram is to the heart and, like the electrocardiogram, it can give a definite diagnosis, a suggestive diagnosis, or it can be abso-

*Presented at the Seventy-first Annual Meeting of the Louisiana State Medical Society, New Orleans, May 9, 1951.

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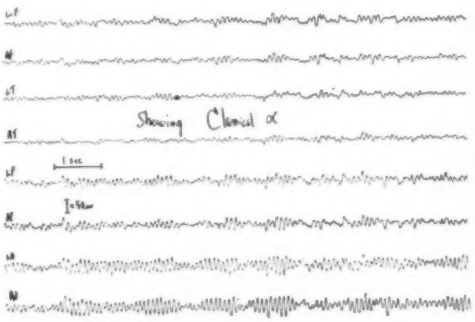
lutely normal in the presence of gross pathology of the organ, or it can be mildly to moderately abnormal in a so-called normal person. All of you, doubtless, accept without condemnation of the technique the fact that the electrocardiogram does not diagnose a valvular stenosis, yet you would never fail to employ the electrocardiogram when it was readily available in the diagnostic work-up of a cardiac case. The electroencephalogram should be regarded in the same way. In many instances, the electroencephalogram will give you an absolute, incontrovertible diagnosis, e.g., in coma, but in most instances the information it will furnish will be ancillary. For this latter reason, the electroencephalogram is best read in clinical terms by one able to relate the electroencephalographic findings to the total diagnostic picture. The electroencephalographer, then, should be a clinically experienced neuropsychiatrist or neurologist. Perhaps one of the simplest analogies to offer in support of that contention is the analogy of the chest x-ray showing an infiltrative, pulmonary shadow—the clinically informed New Orleans radiologist finding such in a native Orleanian who has not left the city since birth is not likely to entertain coccidioidosis as part of his differential diagnosis, although the x-ray findings can be identical with those of tuberculosis. The average busy physician who refers his patient to a radiologist expects a diagnostic impression in report with the radiologist being possessed of specialized, clinical knowledge about his field. The busy physician should expect the same of the electroencephalographer. The obvious corollary, however, is that the electroencephalographer must be supplied with clinical data by the referring physician in order to give a clinical reading. In this latter regard, an English worker, Turner,[1] has said, "Without adequate consideration of the history and findings on clinical examination, it (the EEG) is largely valueless and may be misleading".

Now, a word about the electroencephalogram instrument itself and the manner of making a recording. The instrument is simply a tremendously powerful amplifier which picks up at the scalp surface the minute electrical discharges of the brain via small solder electrodes fastened to the scalp with bentonite paste and lead to the instrument by fine copper wires. The instrument amplifies the activity from 5 to 50 million times and provides that the activity picked up from each electrode, or lead, as it is called, is put through an ink-writing voltmeter which records the impulse variations on a sheet of paper continuously moving at 3 cm/second. Each recorded line on the paper is termed a channel, in reference to the distinct circuit through which the activity has been amplified. A truly adequate instrument has 8 such channels thus enabling one to view the major areas of the brain in simultaneous function. Since the homologous brain areas display practically identical patterns under normal conditions and dissimilar patterns under abnormal conditions, the electrodes are placed on the skull in a symmetric manner: the left and right frontal in identical positions, the left and right temporal similarly, the parietals similarly, the occipitals, and so on. Since the largest instrument built at present has only 8 channels, and the anterior portions of the temporal lobes have particular importance in psychomotor epilepsy as opposed to the posterior aspects, we run two patterns, the frontals, mid-temporals, parietals and occipitals as one simultaneous group, and the frontals, anterior temporals, mid-temporals and parietals as the second group. By virtue of selector switches on the instrument, the brain is constantly scanned, as it were, over the 10 leads throughout a recording. The ideal EEG machine should have all 10 leads visible at once with an extra channel for the ECG. The reason for recording the ECG is that it is a common artifact in the brain recording and the QRS complex can occasionally be mistaken for a spike, the electroencephalographic result of a paroxysmal potential burst in the brain, which is a grossly abnormal finding. At present, we simply run a strip of ECG in a normal channel whenever we need it for reference.

The patient is handled very similarly to the ECG patient. The electrodes are applied with the patient sitting up on a bed and he is asked to recline with eyes closed when all are applied. Ideally, given a patient of sufficient IQ, he is asked to maintain an attitude of alert expectancy but to avoid active cerebration and, above all, to keep still, to relax and try to sleep because muscle tension and movement artifacts tend to obscure the brain's electrical activity. Since sleep tends to bring out pathological activity, every effort is made to get the patient to sleep and in most laboratories patients are routinely given a small dose of short-acting barbiturate or similar sedative-hypnotic. The average recording takes about thirty minutes. When the occasion demands, we put on additional electrodes more precisely to localize a lesion and may run an hour or more. Since the sedative-hypnotics and anticonvulsants, taken routinely, produce a fast artifact in the EEG, the referring physician should interdict such medication about thirty-six hours prior to the EEG if there be no likelihood of status epilepticus developing. This is especially important with the anticonvulsants for they may not only introduce a fast artifact but may suppress the very paroxysmal activity for which we are looking.

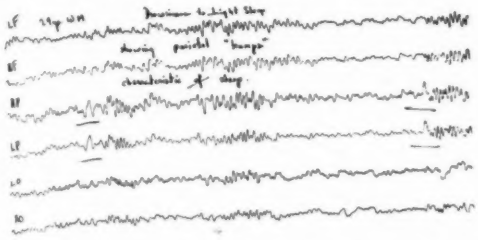
On occasion, and only with the approval of the referring physician, we may administer intravenous metrazol to a patient in the effort to activate a paroxysmal dysrhythmia when there is reason to believe that the patient's brain is subject to such paroxysmal outbursts although his interseizure EEG's are negative. In every case, however, where the patient can coöperate, we hyperventilate the patient for from three to five minutes as alkalosis tends to activate epileptic activity. When that procedure is not fruitful, we often have the patient open and close his eyes in an effort to activate pathologic activity, particularly petit mal. This is particularly true of children with true petit mal. This is a good point to emphasize the fact that the EEG of a known epileptic may be perfectly normal during interseizue periods, and an apparently normal person with no seizure history may display, within certain limits, an epileptoid or definitely epileptic record. It is for those reasons that EEG's are usually reported in statistical terms. An abnormal pattern, depending on its degree, is said to be, going from mild to severe, "consistent with but not suggestive of convulsive disorder", "suggestive of convulsive disorder", and "suggestive" is then qualified up to "very strongly suggestive". So, too, for brain damage where the most definite one can statistically be is to say that the most abnormal pattern is "presumptive evidence" of brain damage. In the absence of a history and neurological findings, that is the way a brain tumor is reported.

In the normal brain and some abnormal brains, recording with the eyes closed since visual activity disrupts the cerebral patterns, the basic wave form arises principally from the parieto-occipital regions and is of sinusoidal form with an amplitude ranging from 30 to 70 microvolts and a normal frequency range of 8.5 through 12/second. (Figure 1). The predominant

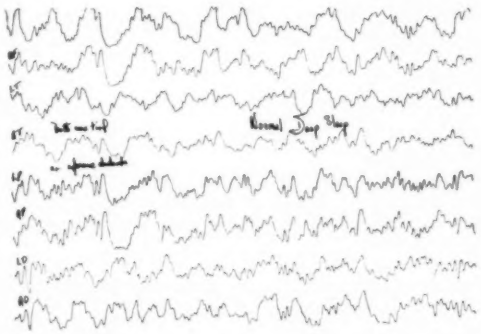


normal frequency is 10/second. These waves are called alpha waves. In the frontal and temporal regions, irregular low voltage activity arises of from 17 to 35/second in frequency which is termed beta activity. In the adult waking record, irregular extremely slow activity having a frequency of 0.5 to 3/second is called delta, or S3, activity and is grossly abnormal. It is, however, normal for the infant. The patterns of children are slower than adults' and the child may not achieve his totally adult frequencies until he is nine-

teen. Any trains of activity in the range of 4 thru 7/second in the waking adult are abnormal and the activity is referred to as S2, or theta, activity, moderately to very slow. With normal sleep in the adult, the voltage drops markedly in all leads. In drowsiness, the record flattens, as we say, and then gives way to a rhythmic slow picture, in light sleep punctuated here and there by roughly one second trains of 14-16/second spindle-shaped waves termed sleep spindles. (Figure 2). With deep



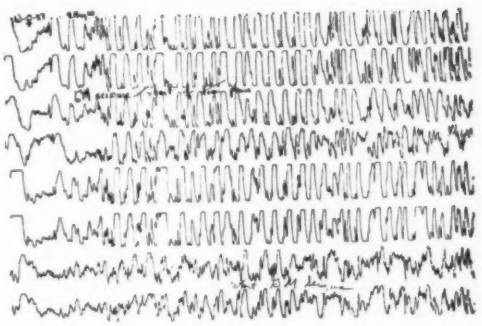
sleep, the entire record becomes even slower and there is a rhythmic rise and fall of the large slow waves, (Figure 3) and the sleep



spindles decrease or practically disappear.

In general the abnormalities of the waking and sleeping patterns which indicate pathology are the presence of excessive fast or slow activity, generally or focally, lack of symmetry of amplitude in homologous leads, and, preëminently, paroxysmal activity such as spikes, spike and dome, delta waves, etc., occurring generally or focally. (Figure 4).

The EEG, then, finds its use in the diagnosis of the epilepsies, in brain damage from any cause, and in the diagnosis and location of space-occupying lesions in the



anterior and middle fossae (tumors of the posterior fossa do not ordinarily reveal themselves to the EEG unless indirectly through the delta activity of increased intracranial pressure). The EEG is a useful tool in the etiologic diagnosis of mental deficiency and in spastic children of normal or defective intellect. A subclinical epileptic spastic may be helped by chemotherapy. The EEG should be done in every case of serious, acute head injury for prognosis as well as diagnosis. I hazard the guess that there would be less deaths and punch-drunks in the professional boxing ring if fighters suffering a knockout were not permitted to train or fight until their EEG's were perfectly normal in serial study. The EEG is useful to the ophthalmologist in the diagnosis of strabismus of central origin. It is useful to the orthopedist contemplating a corrective operation on a spastic child. If the child is a subclinical epileptic, the added sensory input from a corrected limb may raise the brain's electrical potentials to the convulsive threshold. Serial EEG may be useful to the obstetrician in pre-eclamptic cases. Because of its tremendous amplifying power, the EEG easily picks up the fetal heart through the mother's abdomen after the first trimester so that it is a quick means of diagnosing intrauterine death. Although it has little practical value, it can be used similarly to diagnose twins and triplets. The EEG can be a considerable aid to the otolaryngologist in cases of hysterical or simulated deafness. In ophthalmology, since porencephaly not uncommonly follows vascular or traumatic lesions in the occipi-

tal lobes, and since it may give the first hint of an occipital lobe tumor, an EEG should be done in all cases of homonymous hemianopsia. In general, the EEG has no established value in the diagnosis of the functional psychiatric illnesses, neurotic or psychotic, nor is there any characteristic EEG pattern in the psychopath. It is useful, however, in neuropsychiatry in distinguishing organic behavior disorders from functional. A child or adult may not have seizures and yet have incessantly repeated bursts of paroxysmal dysrhythmia in his brain, the so-called subclinical or larval epilepsy, which grossly interferes with his personality function and adjustment and may produce deviant behavior. In the organic psychoses of degenerative type the EEG commonly displays no diagnostically specific pattern nor does it diagnose cortical atrophy or absence from any cause. In acute diffuse cerebral lesions the EEG is of little etiologic diagnostic value for the extremely slow activity which results simply indicates stupor or coma whether on the basis of highly increased intracranial pressure, encephalitis, diabetic coma, or meningitis, etc.

In the nonepileptic, post-traumatic case, the EEG is of little or no value unless serial studies are done which show a change of pattern toward or away from normality, or unless one has a pre-injury EEG with which to compare the post-traumatic studies. The exception to this is where there has been focal brain injury and a spike, or slow wave and spike, focus, or focal depression of sleep spindles appears on the EEG.

The EEG finds its principal use in the diagnosis and prognosis of the convulsive disorders; therefore, it would seem desirable to discuss those disorders in relation to electroencephalography in some detail.

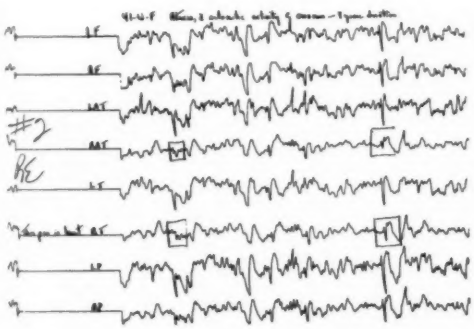
The common, contemporary, clinical classification of the convulsive disorders is anachronistic in that it has not kept pace with the electropathophysiologic findings of the laboratory. Hence, any physician attempting to treat a convulsive disorder in terms of so general a diagnostic classification as grand mal, petit mal or psychomotor epilepsy, without electroencephalographic assistance, is doing himself and his patient an unwitting disservice. I do not include Jacksonian seizures for, obviously, a true Jacksonian seizure identifies itself clinically to the competent eye. A venerable colleague might interpose here that after fifty years of practice he believes he well knows a grand mal convulsion when he sees one. Surely he does, but he can not in a significant and, therefore, in a scientifically prohibitive number of grand mal convulsions, tell by observation and history alone whether they are idiopathic or symptomatic, a vitally important distinction because of the incidence of mortality and the chance of successful surgery in the latter. Wesley Watson,[2] for example, reported that in a series of 279 craniocerebral injury cases of combat origin, 41 per cent of those who developed post-traumatic epilepsy had generalized seizures without focal onset or focal aura. Similarly, we are all familiar with the fact that a generalized convulsion is not uncommonly the first sign of a brain tumor, which, of course, is almost invariably focal. The EEG is the only practical means at present to make a dependable distinction between the principal forms of grand mal.

In general, clinicians talk of the convulsive disorders as idiopathic or symptomatic grand mal (or major epilepsy), focal motor seizures not going on to grand mal, focal sensory seizures, psychomotor epilepsy, and petit mal; and break down petit mal, into pyknoepilepsy or classical petit mal, akinetic epilepsy, and the myoclonic jerk. Recently, Lennox[3] has suggested a fourth type of so-called petit mal; namely, the massive myoclonic jerk. From the standpoint of pathogenesis, however, as reflected in the electrical activity of the variously morbid brains, these clinical categories are not clear cut. Further, treatment directed against a large clinical grouping such as petit mal, diagnostically covering all little seizures, may be a panacea for the one case and poison for the next because what appears clinically as the "Little Sickness" may be any

one of a variety of pathologically distinct disorders. Unhappily, electroencephalography has no uniform, accepted system of clinically correlated classification and terminology worked out as yet which it can present to the clinician, but it *can* tell him what type of electrical dysrhythmia he is dealing with and, ergo, aid in planning the therapeutic course.

Let us now examine the principal clinical types of convulsive disorder and their most important electroencephalographic aspects. We have already mentioned the problem of distinguishing idiopathic and symptomatic grand mal. Of all the EEG patterns indicative of major epilepsy to the electroencephalographer *interpreting the records in terms of adequately supplied clinical information,* almost every one gives him some indication of its etiology—be it brain tumor, focal cortical laceration scar, shrunken gyrus, old encephalitis, birth injury, cerebrovascular accident, or idiopathic epilepsy. The importance of these etiologic indications to useful EEG-reporting with consequent appropriate therapeutic management is too obvious to bear elaboration.

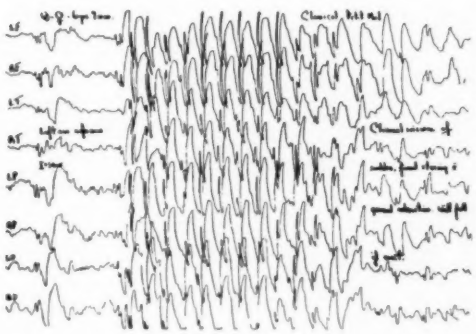
In psychomotor epilepsy, (Figure 5) the



EEG is a diagnostic necessity because of the occasional amenability of one of the two known forms of this disorder to surgical intervention. The two forms of psychomotor epilepsy are, so far, clinically indistinguishable. On the EEG, one form shows a characteristic, generalized pattern of flat-topped waves and a low threshold for the production by metrazol-photic stimulation of a myoclonic spike. The other form shows a precise anterior temporal lobe focus in the EEG, either unilateral or bilateral, and a normally high or higher than normal myoclonic threshold to metrazol-photic stimulation. It is this latter type with the temporal spike focus that has been the subject of surgery at the hands primarily of Penfield, Bailey, and Morris. The EEG also serves prognostically in this latter type postoperatively to determine the continued presence or absence of temporal or paratemporal paroxysmal activity.

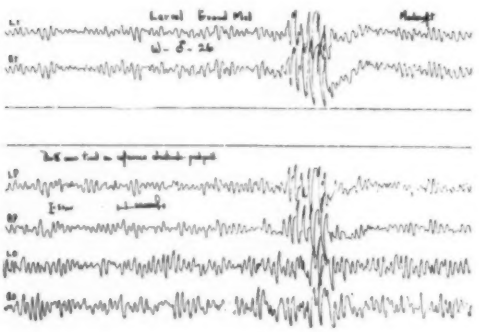
Another diagnostic function the EEG serves in psychomotor epilepsy is in differentiating some psychomotor syndromes from schizophrenia. Prolonged psychomotor attacks of weeks', months', or years' duration can simulate schizophrenia. This is particularly well to remember in dealing with what are termed in the neuropsychiatric ward vernacular "ten day schizophrenics". Henri Gasteau,[4] working at the Neurological Institute in Montreal, has even suggested in a very recent communication that there is evidence for a common diencephalic basis for low myoclonic threshold psychomotor epilepsy and schizophrenia so that the confusion of diagnoses may have pathogenic justification.

Going now to petit mal (Figure 6), as



the term is commonly used, we arrive at a point where it can be dogmatically stated that the clinician using only the art of diagnosis can arrive at a correct diagnosis and appropriate therapy only through good fortune or trial and error. Fortune favors him, also, if he can give the family a valid prognosis. The reason for this is not complex. There are at least four electroen-

cephalographically distinct types of minor seizures which can all look alike clinically but are etiologically and pathologically quite different. There is pyknoepilepsy, characterized on EEG by the 3/second wave and spike, which is what most electroencephalographers are referring to when they use the term petit mal, and which is diencephalic in origin. (Figure 7) There



is the minor motor seizure, as Wilkins[5] terms it, characterized on EEG by *larval* grand mal activity, that is, bursts of grand mal paroxysms in the brain of insufficient duration or force to produce peripheral tonic-clonic convulsive behavior but adequate to give the child or adult a momentary clouding of consciousness, or dullness of comprehension as an epileptic equivalent, once so-called by Putnam and Merritt,[6] or an "absence", as the French say. (Figure

8) There is the petit mal variant minor seizure characterized on EEG by 2/second spike and distorted wave usually cortically focal and apparently not of diencephalic origin, and invariably associated in the same patient sooner or later with major convulsive seizures. Lastly, there are those mild psychomotor seizures characterized by fleeting disorder of perception and awareness which are often clinically confused with petit mal.

All these little seizures described are physiologically different and their optimal treatment is, therefore, different. As time does not permit the enumeration of all these differences, let us use true petit mal (or pyknoepilepsy) and the petit mal variant as examples to contrast. Their little seizures are clinically indistinguishable but their physiologic characteristics are almost antipodal—true petit mal is usually easily made to appear on EEG in sleep and by adequate hyperventilation. Petit mal variant is better seen in a waking record and is either unaffected by hyperventilation or suppressed by it. True petit mal can be made to appear in the EEG by inducing hypoglycemia; petit mal variant cannot. True petit mal can often be made to appear in the EEG by having the patient open and close the eyes; not so petit mal variant. True petit mal is usually generalized in the EEG, whereas the petit mal variant is usually focal. Petit mal commonly exists alone, the petit mal variant usually with, or invariably sooner or later in association with grand mal. In true petit mal the intelligence is only indirectly and temporarily affected. In the petit mal variant definite mental deficiency is the rule. Tridione is the drug of choice, at present, for true petit mal; whereas it often makes the petit mal variant worse. The hydantoins are the drugs of choice for the petit mal variant; whereas they often make true petit mal worse. Lastly, and very importantly, when the clinician must give the members of a family a prognostic answer to their inevitable question, he who knows that his patient has a classical 3/second wave and spike petit mal can confidently reply that the patient usually outgrows it in his early twenties or before, thus, subsequently adding to a reputation for medical wisdom as time confirms the happy prognosis. When the physician knows his patient to have the petit mal variant pattern, he knows that the

prognosis for complete recovery is poor into, if not through, the mature years and he can so inform the family, regretfully, but with assurance of accuracy.

Such a description of two antithetical convulsive entities which are so easily confused diagnostically with one another without the aid of electroencephalography, exemplifies the importance of EEG to the complete diagnosis of cerebral dysfunction. It may give a final and definite answer or be of no aid whatsoever, and in that way it is closely analogous to the electrocardiogram, but, like the ECG, it should always be employed as an adjunctive diagnostic aid when there is suspected or apparent abnormality of the organ the function of which it reflects in bioelectric terms. As stated in the opening words of this paper, the EEG is effectively to the brain what the ECG is to the heart.

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Medical and Surgical Journal

Established 1844

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News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

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SCHEME TO HOSPITALIZE CERTAIN BENEFICIARIES OF THE FEDERAL SECURITY ADMINISTRATION

At various times in the recent past it has been stated that if the Socialists in the administration failed to enact the compulsory sickness insurance program and the state medicine it implies that they would proceed to atempt to secure the adoption of their entire plan by piecemeal legislation. What appears to be such a plan is the recent proposal of Oscar Ewing, the Federal Security Administrator, to provide hospitalization up to sixty days a year for persons, 65 and older, and their dependents who are entitled to Social Security cash benefits, regardless of whether they are actually receiving such benefits or not. Such hospitalization is to be paid for out of Social Security funds now in hand, and expected to be in hand, from payroll deductions already in operation.

The deduction at the present time is 1½ per cent each from the employer and employee. This is scheduled to increase, so that by 1970 the combined percentage will be 6½ per cent. Several years ago, $9,000,000,000 was paid into the Federal Treasury as a sort of loan from the Federal Security Agency, and the daily press has recently reported that another $7,000,000,000 has accumulated at the present time. Funds in these astronomical figures have piled up just since 1933. Such are the powers of taxation!

The operation of the scheme is briefly as follows:

The persons covered would be all persons 65 and over, and their dependents who are entitled to Social Security cash benefits regardless of whether they are actually receiving benefits, also widows under 65 and dependent children, and any other survivors who are eligible for Social Security benefits under the existing law. The Federal Security Administration would regard this hospitalization as the insured right of eligibles. Because of the lack of hospital space at certain times or in certain areas not all applicants could be promised care at a given time. The program would not cover tuberculosis, or mental patients, or most chronic patients. Rest homes and domiciliary homes could not participate.

At the national level, the Federal Security Administrator would set the policy; the Federal Hospital Council would advise him but would have no veto power. The Federal Security Administration would deal with State governments, and State licensed hospitals would be eligible. If the State did not participate, the Administration would deal directly with the hospital.

Sixty days' hospital service would be provided in any one year. Hospitals are to be paid for services, drugs, and appliances which the hospital customarily furnishes to its bed patients. Specifically excluded

are medical care, except that generally furnished as an essental part of hospital care for bed patients. The payments to the hospital would be determined on a mutually agreed basis, which would reimburse the hospital for its reasonable costs incurred for all bed patients occupying semiprivate accommodations in such hospitals. It is anticipated that the program could not be put into operation until 1953, just after the presidential election, and by that time would cover approximately 7,000,000 people. Total cost is estimated as being $200,000,000, more or less, the first year. This is ingeniously arrived at as follows: It is stated that hospitalization in general non-Federal hospitals for the population of the United States, all ages, averages approximately 1 day per person per year. The proposal anticipates a possible 2.5 days of hospitalization per year for the eligibles under this plan. The average cost per day in the type of hospital care anticpated is assumed to be $15. The anticipated $200,000,000 cost the first year could be absorbed out of the existing tax on wages. This amount is expected to be less than 0.2 of 1 per cent of the total taxable payroll for the year 1953, which is estimated as being $141,000,000,000.

In presenting a plea for the adoption of this program the author states that this group of 7,000,000 persons and their dependents, 65 years and older, are people who as a whole need much more than the average amount of hospitalization, they have less than average income with which to meet the costs of hospitalization, and much less than the average opportunity to obtain private insurance.

This program of tax supported sickness insurance enacted into law would expand like an accordion. In a matter of months the age limits would be lowered and the benefits increased, the tax increased, and the whole program of socialized medicine would be on the highway to enactment. The sequence of events by which this would be accomplished is easy to foresee. Politicians would vie with each other in promising and voting for more and more "free" benefits under the so-called insurance system. The public does not realize that there is nothing free; that the Government has no money. The only money it can give away is that which it takes from the taxpayers; and that the "free" part represents somebody's toil. If ever there was an attempt to sell a Trojan horse scheme to the American medical public, this is it.

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The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

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C. Grenes Cole, M.D., Chairman; W. H. Gillentine, M.D.; both of New Orleans; J. E. Knighton, M.D. Shreveport.

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Committees:

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History of LSMS:

Rudolph Matas, M.D., Chairman; D. N. Silverman, M.D.; Isidore Cohn, M.D.; all of New Orleans.

Hospitals:

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Lectures for Colored Physicians:

Daniel J. Murphy, M.D., Chairman; J. D. Rives, M.D., Conrad G. Collins, M.D.; all of New Orleans; P. D. Abramson, M.D.; Chas. R. Gowen, M.D.; both of Shreveport.

Medical Indigency:

Jules M. Davidson, M.D., Chairman; New Orleans; H. Guy Riche, Jr., M.D., Baton Rouge; Chas. Russell Brownell, M.D., Morgan City; T. B. Sellers, M.D., New Orleans; Douglas L. Kerlin, M.D., Shreveport.

National Emergency Medical Service (Civil Defense):

Charles B. Odom, M.D., Chairman; Val H. Fuchs, M.D.; both of New Orleans; Rhett McMahon, M.D., Baton Rouge; Willard A. Ellender, M.D., Houma; Jared Y. Garber, M.D., Lake Charles; C. P. Herrington, M.D., Alexandria; Henson S. Coon, M.D., Monroe; William J. Norfleet, M. D., Shreveport; E. H. Byrd, M. D., Leesville; E. P. Breaux, M.D., Lafayette; F. C. Shute, M.D., Opelousas.

Nominations:

(Committees to be elected at 1952 Annual Meeting).

Pension Plan for LSMS Office Employees, in re Investigation of:

L. A. Monte, M.D., Chairman; Eugene B. Vickery, M.D.; Sam Hobson, M.D.; all of New Orleans.

Procurement and Assignment (La. Advisory Committee to Selective Service):

J. Kelly Stone, M.D., Chairman; H. Ashton Thomas, M.D.; both of New Orleans; Guy R. Jones, M.D., Lockport; M. D. Hargrove, M.D., Shreveport; H. H. Cutler, M.D., Monroe; Rhett G. McMahon, M.D., Baton Rouge; G. E. Barham, M.D., Lake Charles; M. B. Pearce, M.D., Alexandria.

Public Health of the State of Louisiana:

A. V. Friedrichs, M.D., Chairman; Roy B. Harrison, M.D., Nicholas J. Chetta, M.D.; all of New Orleans; C. J. Schexnaildre, Jr., M.D., Franklin; W. J. Sandidge, M.D., Shreveport; W. L. Bendel, M.D., Monroe; Carlos Alessi, M.D., Hammond; R. G. Holcombe, Jr., M.D., Lake Charles; Roy W. Wright, M.D., Pineville.

Rural and Urban Health:

J. P. Sanders, M.D., Chairman, Shreveport; Guy R. Jones, M.D., Vice-chairman, Lockport; Geo. H. Hauser, M.D.; J. J. Irwin, M.D.; both of New Orleans; James E. McConnell, M.D., Monroe; M. C. Wiginton, M.D., Hammond; Arthur Vidrine, M.D., Ville Platte; J. A. White, Jr., M.D., Alexandria.

Second District Med. Soc. & OPMS, in re Investigation of Membership In:

E. L. Leckert, M.D., Chairman; A. V. Friedrichs, M.D.; Roy B. Harrison, M.D., all of New Orleans.

Woman's Auxiliary (Advisory Committee):

Roy B. Harrison, M.D., Chairman; H. Theodore Simon, M.D., Vice-chairman; Edwin Guidry, M.D.; all of New Orleans.

PLANNING BOARD IN RE SURVEY OF FACILITIES AND PERSONNEL FOR MEDICAL CARE

Val H. Fuchs, M.D., Chairman, New Orleans; J. Y. Garber, M.D., Vice-chairman, Lake Charles; Alton Ochsner, M.D., New Orleans; Felix A. Planche, M.D., New Orleans; J. W. Faulk, M.D., Crowley; Paul D. Abramson, M.D., Shreveport; P. A. Donaldson, M.D., Reserve.

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M. M. Hattaway, M.D., Chairman; C. F. Bellone, M.D., Vice-chairman; Theodore F. Kirn, M.D.; all of New Orleans; Leo J. Kerne, Thibodaux; T. B. Tooke, Jr., M.D., Shreveport; T. A. Dekle, M.D., Jonesboro; Daniel J. Fourrier, M.D., Baton Rouge; Max M. Miller, M.D., Lake Charles; O. B. Owens, M.D., Alexandria; P. H. Jones, M.D., New Orleans (Editor, NOMSJ) ex-officio member; C. Grenes Cole, M.D., New Orleans (Sec. Treas. State Society) ex-officio member; Edwin L. Zander, M.D., New Orleans (Pres. State Society) ex-officio member.

EXECUTIVE COMMITTEE

Edwin L. Zander, M.D., New Orleans—President..

W. E. Barker, Jr., M.D., Plaquemine—President-elect.

Charles B. Odom, M.D., 1707 Pere Marquette Bldg., New Orleans—First Vice-President.

H. Whitney Boggs, M.D., 803 Jordan St., Shreveport—Second Vice-President.

W. Robyn Hardy, M.D., 601 Maison Blanche Bldg., New Orleans—Third Vice-President.

George W. Wright, M.D., Monroe—Past President.

A. V. Friedrichs, M.D., 840 Maison Blanche Bldg., New Orleans—Chairman, House of Delegates.

T. Benton Ayo, M.D., Raceland—Vice-Chairman, House of Delegates.

C. Grenes Cole, M.D., 1430 Tulane Avenue, New Orleans—Secretary-Treasurer.

H. Ashton Thomas, M.D., 922 Richards Bldg., New Orleans—Councilor, First District.

Joseph S. Kopfler, M.D., Kenner—Councilor, Second District.

Guy R. Jones, M.D., Lockport—Councilor, Third District.

Paul D. Abramson, M.D., 1130 Louisiana Avenue, Shreveport—Councilor, Fourth District.

C. Prentice Gray, Jr., M.D., Monroe—Councilor, Fifth District.

Arthur D. Long, M.D., 1140 Perkins Road, Baton Rouge—Councilor, Sixth District.

J. W. Faulk, M.D., Crowley—Councilor, Seventh District.

H. H. Hardy, M.D., Alexandria — Councilor, Eighth District.

REPORT OF DELEGATES 1951 AMA MEETING

To the President and Members
of the Executive Committee
Louisiana State Medical Society
Gentlemen:

The following report covers the proceedings of the Annual Meeting of the House of Delegates of the A. M. A., held at Atlantic City, N. J., June 11th through the 15th, 1951.

The House was called to order promptly at 10 a. m., June 11th, by the speaker, Dr. F. F. Borzell.

It was gratifying to see that 200 out of a possible 201 delegates, were seated during the meeting. For the first time, two members of the newly formed "Student American Medical Association" were seated in the House as Delegates from their organization. It may interest you to know that 40 medical schools so far ratified the constitution out of the 47 schools attending the organization meeting. It is felt that ultimately all medical schools will become affiliated.

It was voted to make the immediate five past

presidents automatically members of the House with full powers of Delegates.

The rule in regard to Delegates was changed. In the past, once a delegate was seated, he could not be replaced. The rule now permits the Alternate to be seated when conditions will not permit the Delegate to attend the sessions, but once the Alternate has been seated, he must continue to serve throughout the rest of the session, and no further replacements will be permitted.

The Board of Trustees established rules for exemption from membership dues, and they were approved by the House. These rules are:

(1) Members who have retired from the practice of medicine, provided they are also excused from the payment of dues, in full or in part, by their component societies and constituent associations. (This will necessitate an amendment to the By-Laws, Chapter II, Section 3, and has been brought to the attention of the Committee on Constitution and By-Laws.)

(2) Members over 70 years of age, regardless of whether or not they are in practice and regardless of local dues exemption.

(3) Members for whom the payment of dues constitutes a financial hardship and who are also excused from the payment, in full or in part, of component and constituent society dues for the same season. In each case, notification of exemption for financial hardship should be made to the American Medical Association by the Secretary of the county and state medical society.

(4) Interns and residents not more than five years after graduation from medical school, except that time spent in military service may be excluded in calculating the five year limit.

(5) Members who enter military service prior to July 1 of any year are exempted from one half of the year's dues and subsequently during service from full dues.

The question of delinquent dues was referred back to the Board of Trustees for study and decision. The Committee itself disapproved waiving of the 1950 dues.

The special Committee of the Board of Trustees to study the Twelve Point Program was empowered to formulate a "statement of policy" for their future guidance.

State societies are to be advised to be careful that the dates of their meetings do not conflict with the dates of local or national elections.

A resolution was unanimously adopted on the death of Dr. Roy W. Fouts, a former Speaker of the House.

The contract of Whitaker and Baxter terminates this year, and the Board of Trustees decided not to renew it. On recommendation of the House, the Board of Trustees decided to retain this firm to work with the Coordinating Committee.

Dr. Henderson made it very clear that any funds contributed to the A. M. A. Educational Fund can be earmarked for a particular medical school by the donor. The cost of dispensing these funds is absorbed by the Board of Trustees. The Woman's Auxiliary presented the fund with a check for $10,000, and the American Academy of Radiology presented a check for $2000 at this meeting.

The Committee on Blood Banks distributed a comprehensive report in bulletin form, and they strongly advise against mass typing of blood.

On the last day the Election of Officers was held, with the following results:

1. President-Elect—Dr. Louis H. Bauer.
2. Vice-President—Dr. O. S. Hunter.
3. Secretary—Dr. George F. Lull (Re-elected).
4. Treasurer—Dr. J. J. Moore (Re-elected).
5. Speaker of the House—Dr. F. F. Borzell (Re-elected).
6. Vice-Speaker of the House—Dr. James Reuling (Re-elected).

Dr. Walter B. Martin was re-elected to the Board of Trustees for a five-year term, and Dr. David B. Allman was elected to serve out the unexpired term of Dr. Bauer:

Dr. Frank Murphy was elected to the Council of Medical Education and Hospitals to succeed Dr. W. S. Middleton, who refused further appointment.

At the Conference of Presidents and other officers of state medical associations held on Sunday, June 10, we were rewarded with four talks that could not be equalled on any one program:

1. A PHYSICIAN VIEWS MEDICINE—by Dr. W. Andrew Bunten, Cheyenne, Wyo.
2. AN EDITOR VIEWS MEDICINE—by Edwin F. Abels, Lawrence, Kansas.
3. A CLERGYMAN VIEWS MEDICINE—by Most Reverend John J. Wright, D.D., Ph.D., Roman Catholic Bishop, Worcester, Mass.
4. A LEGISLATOR VIEWS MEDICINE—by Hon. Richard M. Nixon, Whittier, Calif.

At the dinner for the Delegates on Monday night, June 11, Mr. Dan Beck, Executive- Vice-President of the International Brotherhood of Teamsters, A. F. of L., delivered an excellent address entitled, "Government Medicine — Danger Ahead." This address was carried over two nation wide hookups, and it is unfortunate that every practicing physician could not hear this talk.

On Tuesday night, June 12, Dr. John W. Cline was installed as President, and since he is an excellent speaker, there is no doubt that his inspiring talk was well received on the nation wide broadcast.

Your delegates were pleased to note that both the President of the State Society, Dr. Edwin L. Zander, and the Secretary-Treasurer, Dr. C. Grenes Cole, attended every session of the House and in addition, some of the Committee meetings.

Your delegates want you to know that we appreciate the honor of representing the membership of our great State Society, and we pledge ourselves

again to work for the interest of our membership as a whole and will always act according to your instructions to the best of our ability.

The final tabulation was not in when we left, but it was thought that the registration would reach or exceed 13,000 doctors.

JAMES Q. GRAVES, Delegate
VAL. H. FUCHS, Delegate

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LOUISIANA PHYSICIANS SERVICE

On May 7, 1951, the House of Delegates of the Louisiana State Medical Society advised that the Louisiana Physicians Service should separate entirely from joint operation with Louisiana Hospital Service, and that it should proceed to sell surgical, medical, and hospital insurance throughout the State. This action was taken after the House of Delegates had received the report of the committee appointed by the President to advise the House of Delegates in regard to certain matters presented to it in connection with the operation of these two insurance companies. Following this, certain activities of Louisiana Hospital Service were observed which the Louisiana Physicians Service considered to be prejudicial to the interests of the Louisiana Physicians Service and in opposition to the best insurance practice.

Suit was filed in the Civil District Court in East Baton Rouge Parish by Louisiana Physicians Service, seeking an injunction against Louisiana Hospital Service, and this was granted and made permanent after a series of hearings by Judge Spaht. In addition, a plea for a hearing before the insurance commissioner was entered in the office of the Secretary of State by Louisiana Physicians Service, and on July 27, 1951, a decision was rendered by the Honorable Wade O. Martin, Jr., Secretary of State and ex-officio Insurance Commissioner, in favor of Louisiana Physicians Service. This decision suspended the certificate of authority of Louisiana Hospital Service, Inc., for a period of sixty days from the date of the order. Suspension prevents them from undertaking any new business during the period of the action of the order, but allows them to operate and service old contracts.

The rights of Louisiana Physicians Service have by this means been protected and the operation of the business made more secure. The legitimate interests of the Louisiana State Medical Society have been served expeditiously. Other facts concerning this situation will appear in the next issue of the Journal.

DR. RALPH E. KING GIVEN THE AXSON-CHOPPIN AWARD

Dr. Ralph E. King, state senator from Winnsboro, was presented the Axson-Choppin Award at the annual meeting of the Louisiana Association of Public Health Workers which was held recently in Alexandria.

This award, which honors the memory of the first and second state health officers in Louisiana, is presented annually by the association to someone outside the field of public health who has rendered valuable service to the cause of public health in the state. A large plaque was given to the winner.

VETERANS ADMINISTRATION, SHREVEPORT

The Veterans Administration Center is urgently in need of a doctor to fill a vacancy on their Adjudication Rating Board, as Medical Officer.

The entrance salary for this position is $6400.00 per annum. Upon the completion of every 18 months of service, the basic salary is increased $200.00 per year up to a maximum salary of $7400.00. The hours of duty presently observed by this installation are from 8:00 A.M. to 4:30 P.M., Monday through Friday.

In a position of this nature, the doctor on the Rating Board will adjudicate the extent of physical disabilities of claimants for the purpose of awarding compensation to persons because of injuries on the job or compensation or pension for service connected or non-service connected disabilities to veterans or surviving dependents.

Anyone interested should contact the Personnel Officer, Veterans Administration Center, 510 E. Stoner Avenue, Shreveport, Louisiana, as soon as possible.

U. S. VITAMIN CORP. BUYS ARLINGTON CHEMICAL CO. COMBINED PLUS NEW FACILITIES WILL ENLARGE SERVICE TO PROFESSIONS

U. S. Vitamin Corporation, New York, N. Y. has announced the purchase of time-honored Arlington Chemical Company of Yonkers, N. Y. The 72,000 square foot plant, together with a large new building to be constructed thereon, will be utilized to enlarge the services of both U. S. Vitamin and Arlington to the medical and pharmaceutical professions.

Steeped in tradition as a manufacturing pharmaceutical house, Arlington has pioneered in the introduction of many highly regarded products since its inception in 1887, almost three quarters of a century ago. A protein preparation first marketed in 1893 was the forerunner of today's widely used protein hydrolysates, of which Arlington's Aminoids is one of the best known.

It is stated that the present and planned new manufacturing and Laboratory facilities, together with an additional score of chemists and other scientists, will lead to the introduction of several new products in the near future.

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LOUISIANA ACADEMY OF GENERAL PRACTICE SEPTEMBER 5-7, 1951

The Louisiana Academy of Dental Practice announces the Fifth Annual Scientific Assembly, to be held at the La Tropicana in Baton Rouge. The following program has been arranged:

WEDNESDAY, SEPTEMBER 5

7:30 P. M. Annual Banquet for the State and the District Officer

THURSDAY, SEPTEMBER 6

8:00 A. M. Registration

9:00 A. M. Meeting of the House of Delegates

12:00 Noon Luncheon for the House of Delegates

1:30 P. M. Opening Exercises
Remarks by the National President
J. P. Sanders, M.D., Shreveport, Louisiana

2:00 P. M. Samuel Fitzsimons Ravenel, M.D., Greensboro, North Carolina
Dean of Southern Pediatric Seminar, Saluda, North Carolina
"Practical Points in Pediatric Practice"

2:45 P. M. R. L. Sanders, M.D., Memphis, Tennessee
Professor of Surgery, University of Tennessee
"Diagnosis and Treatment of Acute Abdomen"

3:30 P. M. Recess and Visit Techanical Exhibits

4:00 P. M. Harry Merrill Murdock, M.D., Townsend, Maryland
Medical Director of the Shepherd, Enoch Pratt Hospital, Associate Professor of Psychiatry, University of Maryland
"Diagnosis and Treatment of Office Psychiatric Problems"

4:30 P. M. Chauncey Mayer, M.D., Chicago
Associate Professor of Medicine, Northwestern University
Attending Staff at Passavant Memorial Hospital, Cook County Hospital, Chicago
Author of *Electrocardiography* of several editions
"Diagnosis of Acute Coronary Thrombosis"

7:30 P. M. Banquet
Introductions
Installation of Officers
Speaker: Mac F. Cahal, M.D., Kansas City, Missouri
Executive Secretary and General Council of American Academy of General Practice
"A Twentieth Century Renaissance in American Medicine"

FRIDAY, SEPTEMBER 7

8:00 A. M. R. L. Sanders, M.D., Memphis, Tennessee
Professor of Surgery, University of Tennessee
"Gall Bladder Disease: Differential Diagnosis and Treatment"

8:45 A. M. Robert Merrill Murdock, M.D., Townsend, Maryland
Medical Director of the Shepherd, Enoch Pratt Hospital
Associate Professor of Psychiatry, University of Maryland
"Office Psychiatry"

9:15 A. M. Recess and Visit Exhibits

10:00 A. M. Chauncey Mayer, M.D., Chicago
Associate Professor of Medicine, Northwestern University
Attending Staff at Passavant Memorial Hospital, Cook County Hospital, Chicago
Author of *Electrocardiography* of several editions
"Diagnosis and Management of Cardiac Emergencies"

10:45 A. M. Moving Picture: San Francisco Convention, Wyeth, Inc.

11:15 A. M. Samuel Fitzsimons Ravenel, M.D., Greensboro, North Carolina
Dean of Southern Pediatric Seminar, Saluda, North Carolina
"Practical Points in Pediatrics"

12:15 P. M. Luncheon
Round Table Discussions
All speakers present

2:30 P. M. Meeting of the House of Delegates
Executive Board Meeting with the

Officers of the State and the District Societies

A speaker for this post-graduate scientific program was sponsored by Wyeth, Incorporated.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

OUACHITA PARISH

The Ouachita Parish Medical Auxiliary held their monthly luncheon at the Bayou DeSiard Country Club on Thursday, June 14, 1951. The guests were: Mrs. Arthur Herold, National President of the Women's Auxiliary to the American Medical Association and Mrs. Gus Street, Past President of the Mississippi State Auxiliary. Mrs. A. G. McHenry, Mrs. R. M. Simonton, Mrs. W. H. Webster and Mrs. Irving Wolff were the hostesses for the luncheon.

There were many members and their guests attending the luncheon. Among those present were: Mrs. A. L. Peters, Mrs. H. S. Coon, Mrs. N. Kalm, Mrs. W. C. Cookston, Jr., Mrs. Joseph Michaud, Mrs. Henry Guerriero, Mrs. W. Carroll Summer, Mrs. D. L. Anderson, Mrs. W. H. Webster, Mrs. Ralph Armstrong, Mrs. D. M. Moore, Mrs. Stanley Mintz, Mrs. B. J. Lacour, Mrs. J. Schonlau, Mrs. Jack Rawls, Mrs. A. Tisdale, Mrs. John Pracher, Mrs. Bernard Soto, Mrs. George Variono, Mrs. J. Walsworth, Mrs. A. G. McHenry, Mrs. Clifford Johnson, Mrs. Phanar Perot, Mrs. C. B. Flinn, Mrs. Ben Cobb, Mrs. Hayden Cutler, Mrs. A. Scott Hamilton, Mrs. Guy Williams, Mrs. E. M. Clark, Mrs. Prentice Grey, Mrs. DeWitt Milam, Mrs. Cyril Yancy, Mrs. Fred Marx, Mrs. J. B. Vaughn, Mrs. Prentice Gray, Jr., Mrs. Ralph Talbot.

The Ouachita Parish Medical Auxiliary invited the public to hear Mr. O. J. Bori, of Vicksburg a statistician speak on Americanism. Mr. Bori's inspiring lecture was entitled "This is Our Problem."

BOOK REVIEWS

Hematology: For Students and Practitioners: By Willis M. Fowler, M. D. Rev. 2d ed. New York, Paul B. Hoeber, Inc., 1949. Illus. pp. 535. Price $8.50.

The author of this book has admirably fulfilled his expressed intention of writing a textbook of hematology to satisfy the requirements of medical students and the practitioner. He makes no attempt to approach the subject from the viewpoint of the trained hematalogist, though they too will find the material presented in a concise, well organized manner. There is no detailed discussion of controversial material except where such knowledge is essential to the interpretation. Hematology is herein presented as essentially a part of internal medicine, with emphasis on the clinical and therapeutic aspects of the various diseases, rather than as a specialty—an approach of great value to the medical student. A definite effort is made to incorporate many of the recent therapeutic advances in this second edition, making it of current value. The style is simple and lucid, and the book on the whole makes very easy reading, so that while there is startlingly new approach to the subject material, it will be a useful book for the student.

GEORGIANA VON LANGERMANN, M. D.

Record of the Past Presidents' Advisory Council of the Louisiana State Medical Society during my Presidency, 1928-50; compiled by Rudolph Matas, M. D. (Manuscript) pp. 259. illus. por.

On Monday, May 7, 1951, Dr. Rudolph Matas, president emeritus of the Past Presidents Advisory Body of the Louisiana State Medical Society, through his successor, Dr. Leon Menville, presented to the House of Delegates of the State Medical Society a record of the activities of the Past Presidents' group during the twenty-two years as president, from organization of the group in 1928, until his retirement from office at the meeting in Baton Rouge in 1950, when he was made president emeritus.

The record, a 250 page, three inch thick quarto volume with dark red imitation morocco scrapbook cover, consists of a collection of original invitations to annual dinners, original menus, newspaper clippings, summaries and minutes of proceedings of each dinner, historical account of the informal organization and formal creation of the advisory body by the House of Delegates in 1931, with portrait and dedication to Dr. Robert Douglas who presented the primary resolution which gave official status to the past presidents as an advisory body, portraits of each president of the Louisiana State Medical Society from 1928-1950, of Dr. Matas and of a few others, biographic sketches and eulogies of deceased members, reprints of several articles written by Dr. Matas during his presidency relative to the Past Presidents, to the state medical society and the medical profession of Louisiana,

and clippings from the 1950 summer edition of the Tulanian.

Dr. Leon Menville, who succeeded Dr. Matas as President of the Past-Presidents' Advisory Council, acting for Dr. Matas in presenting the volume to the Louisiana State Medical Society, suggested that the volume be placed in the Rudolph Matas Medical Library of Tulane University, where it would always be available for use. Presentation to the Matas Library was made by Dr. C. G. Cole, Secretary-Treasurer of the Louisiana State Medical Society, on May 10. This volume forms an invaluable addition to the collection to be found in this library on the Medical history of Louisiana.

Pathologic Physiology: Mechanisms of Disease. Edited by William A. Sodeman, M. D., F. A. C. P. Philadelphia, Penn., W. B. Saunders Company 1950. pp. 808. Price, $11.50.

When one undertakes to review a new book there are several question that he must answer. What need is there for a book of this sort? Are there are not other books in this category that are sufficient for the purposes thereof? Does this book meet the requirements of the need? Is the data presented and the manner of presentation intelligible for the reader for whom it is intended? To offer a proper answer in the affirmative or negative requires of the physician that he be cognizant of the fact the modern structure of medicine rests on the pillars of physiology and biochemistry. He must also know that modern medicine requires an encyclopedic knowledge that the modern physician cannot hope to possess.

Sodeman's book is the collaborative effort of 25 authors. Each one is supposedly authoritative in his own field. How then can one presume to pass judgment on the worth of the many contributions presented in this book. It might be suggested that one with a good background and excellent training in biochemistry and physiology might be chosen to state whether it contains sound modern concepts acceptable to all authorities. But this volume is not intended for such persons. What then about the validity of a review by a practising physician? Can it be said with sincerity that any physician possesses the knowledge of the many and intricate theories and facts, physiologic in nature, that abound in all departments of medicine. This is not a textbook of medicine. Symptoms and signs are treated principally from the viewpoint of nosology. What then becomes necessary for the physician is that he have faith in the trusworthiness of the assembled data. Since the reader cannot test the value of the facts he must be able to attest to the honor and integrity of the authors. This then is a prime necessity. Coupled with this must be the knowledge that the authors are truly informed and able to write with authority. Between research scholars and practising physicians there must be a liaison. This liaison can only be successful if the authors, fully appreciative of the theoretical garnishments are able to present the practical fact—the meat—so that it is obvious, palatable, and digested with reasonable effort, satisfaction and benefit.

The reviewer believes that Sodeman's *Pathologic Physiology* fulfills all of the above requirements. Here is a work by research and practising physicians whose merited reputations are already established; who are keenly aware of the great importance of scientific research and yet cognizant of the needs of the practising physician. The happy blending of theory and fact in the realm of the raison d'etre of symptoms and signs and treatment is eminently achieved. The reviewer feels that every practising physician seriously intent on an understanding of the bases of modern medicine can use this knowledge to great advantage, secure in the knowledge that the facts are factual; the authors authoritative; and the explanations explanatory. The excellent format and index of the book with its fine illustrations, paper and print are niceties to be expected in a welcome, workable addition to our armamentarium. Dr. Sodeman, who needs no introduction, is to be congratulated and commended for the fine company he keeps within the covers of this splendid book.

I. L. Robbins, M. D.

Bronchoesophagology: By Chevalier Jackson, M. D., Sc. D., LL. D., F. A. C. S. and Chevalier L. Jackson, M. D., M. Sc., F. A. C. S., Philadelphia, W. B. Saunders Co., 1950. pp. 366. illus. pl. Price, $12.50.

A narration of some past events, which are not mentioned in the book being reviewed, is appropriate as a tribute to the genius and perseverance of the senior author.

This book is based, as would be any book on bronchoesophagology published today, on the fundamental clinical observations of Dr. Chevalier Jackson, made in the years from about 1890 to 1925 when he was the principal artisan in moulding the science of bronchoesophagology.

Dr. Jackson was a man with a mission, and the turning point in his career was the restriction of his physical activities in 1911 due to pulmonary tuberculosis. This decided him to confine his work to laryngology and to bronchoscopy and esophagoscopy (later he proposed the more appropriate terms bronchology and esophagology). During his convalescence from tuberculosis the hours of enforced bed rest were courageously and stubbornly used to the utmost in writing, collecting notes, and painting illustrations for his first widely read text, *Peroral Endoscopy and Laryngeal Surgery* which appeared in 1915. If Dr. Jackson had not had the misfortune to have had tuberculosis, the

world would have had very probably the misfortune of long delay in the fruition of bronchoesophagology as a science. In his early years and for many years this frail man carried the burden of "him who is the court of last resort", and gave charitably and unstintedly of his life saving ability. No one who had not known him and his work in those days, with the world clamoring for his services, could appreciate the load he carried.

Dr. Chevalier L. Jackson has become distinguished in his own right and has able carried on the work of his illustrious father. Together, they have produced in *Bronchoesophagology* an indispensable reference work for every one interested in bronchoesophagology; familiarity with its contents will help the practitioner of this specialty solve his problems.

Well deserved commendation is due the publishers for the excellence of the entire format of the volume.

H. KEARNEY, M. D.

Cranioplasty: By David L. Reeves, A. B., M. D. Springfield, Ill. Charles C. Thomas, 1950. pp. 119, illus. Price $3.00.

This well illustrated monograph reviews briefly but interestingly the history and technics of cranioplasty, the advantages of the various materials which have been used to repair cranial defects, together with the reasons why most of them have been discarded, the indications for and the possible complications following cranioplasty, and the methods of preparing and applying tantalum plates. Interesting data concerning the author's personal series of 196 cases of cranioplasty are included. Details of the histories and postcranioplasty reactions of 16 of these are described. One hundred twenty-seven of the cases were followed long enough to tabulate the permanent neurologic defects. Convulsive disorders resulted in 20.47 per cent of these; all were cases in which the dura and the cerebral cortex had been penetrated at the time of the injury. The author predicted that a survey of these patients after five years had elapsed postoperatively would reveal an incidence of convusions of 35 to 50 per cent. The commonest neurologic defect was hemiplegia. For those who wish to deal more extensively with certain aspects of cranioplasty than is possible in such a monograph, an excellent bibliography is included. It is evident that this represents avaluable text for neurologists and neurosurgeons.

H. D. KIRGIS, M. D.

Dictionary of Psychoanalysis; ed. by Nandor Fodor and Frank Gaynor, Philosophical Library, New York, 1950. pp. 208. Price, $3.75.

This is a little book of scattered quotations from Freud's numerous papers. It is not a dictionary because so much of it consists of incomplete and uninforming statements. For example, all that is quoted under the heading of phantasy is one isolated statement, "Our phantasy always works on existing patterns". This may be interesting but it hardly does justice to Freud's genius. Some topics are covered fairly completely but in a way that is confusing because dates of references are not given to show how his concepts changed over the many years. Also the book would have had more value if the editors had made the list of his papers complete.

The title is misleading because psychoanalysis is not confined or limited to Freud's contributions.

It is not a good reference book, but it has this value; one can use it to scan the whole territory of Freud's explorations in a short time, and it should encourage the reader to go back and enjoy the original paper.

WALKER THOMPSON, M. D.....

A Primer for Diabetic Patients: By Russell M. Wilder, M. D. Philadelphia, W. B. Saunders, 1950. (Rev. 19th Edition3. Illus. pp. 199. Price, $2.25.

This is the ninth edition of the Primer, with revisions to conform with the therapeutic advancements made since 1946.

The Primer is small enough in size to be carried by the diabetic patient; it presents material to be thought over and understool by the patient under the guidance of his physician. Consisting of nine chapters, the book is concerned with the orientation of the diabetic patient with regard to immediate problems which may arise as well as those to be handled over long periods. It presents the physiology of diabetes, the treatment of diabetes, methods of testing urine, the methods to be used by the patient in handling his insulins, as well as the preventive aspects of the complications of diabetes. Several chapters concentrate on the practical aspects of dietary measures, including substitions and recipes.

The handbook is an excellent guide to be used by diabetics in conjunction with their physician's supervision. Its material could be understood even better if pictorial diagrammatic aids emphasized all the discussions, although the average patient with supervision can handle the material quite adequately.

JOSEPH E. SCHENTHAL, M. D.

Amusing Quotations for Doctors and Patients; edited by Noah D. Fabricant, M. D. New York, Grune & Stratton, Inc., 1950. pp. 149. Price, $3.00.

This collection of clever and amusing quotations on all phases of medicine is arranged in groups

alphabetically by subject. It will prove especially useful to those called upon for public addresses or after dinner talks. The quotations are well chosen, the authors ranging from Marcus Aurelius to Irvin S. Cobb, from Samuel Johnson to Don Marquis. There are few such collections available; this volume will therefore be a welcome addition to the physician's private library as well as to the medical library collection.

MARY LOUISE MARSHALL.

PUBLICATIONS RECEIVED

The Blakiston Co., Phila.: Spatial Vector Electrocardiography, by Robert P. Grant, M. D., and E. Harvey Estes, Jr., M. D.; Practical Clinical Psychiatry, by Edward A. Strecker, M. D., Franklin G. Ebaugh, A. B., M. D., and Jack R. Ewalt, M. D.; Section on Psychopathologic Problems of Childhood, by Leo Kanner, M. D.

Chemical Publishing Co., N. Y.: Science French Course, by C. W. Paget Moffatt, M. A., M. B., (revised by Noel Corcoran B. A. (Com.), B. Sc., M. Sc. Tech.

Modern Medicine, Minneapolis: Modern Medicine Annual—1943 (An annual volume containing the articles which appeared in the twelve issues of Modern Medicine for 1943).

Philosophical Library, Inc., N. Y.: From a Doctor's Heart, by Eugene F. Snyder, M. D.

W. B. Saunders Co., Phila.: Clinical Pediatric Urology, by Meredith Campbell, M. D.

Charles C. Thomas, Springfield, Ill.: The Physiology of the Newborn Infant, by Clement A. Smith, M. D. (2nd Edit.).